REPORTS

OF THE

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

VOLUME VI.

Session of 1905-1906.

EDITOR :
GEORGE CARPENTER, M.D.

Hon. Secretaries:
LONDON :
EDMUND CAUTLEY, M.D. FRANCIS JAFFREY.
PROVINCIAL :
R. H. ANGLIN WHITELOCKE, C.M. (Oxford).

8/3
28/1

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FRANCIS JAFFREY.

OFFICE BEARERS AND MEMBERS OF THE COUNCIL FOR THE SESSION OF 1906-1907.

ELECTED AT THE ANNUAL GENERAL MEETING.

Council (1906-1907).

Those marked thus (*) served on the Council or held office during last session, 1905–6.

A. J. ALLIOTT (Sevenoaks).
*JOSEPH BLUMFELD, M.D.
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S. H. A. LAMBERT, M.B. (Harrow).
PHILIP G. LEE (Cork).
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R. G. McKERRON, M.D. (Aberdeen).
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*R. H. A. WHITELOCKE, C.M. (Oxford).

Honorary Treasurer.

*R. CLEMENT LUCAS, B.S.

Honorary Editor of Reports.

*GEORGE CARPENTER, M.D.

Honorary Secretaries.

LONDON:

*FRANCIS JAFFREY. *J. PORTER PARKINSON, M.D.

PROVINCIAL:

*HENRY SKELDING, B.C. (Bedford).

LIST OF OFFICE-BEARERS SINCE THE FORMATION OF THE SOCIETY IN THE YEAR 1900-1901.

Dates.	Chairmen of Council.	Honorary Secretaries.		Honorary Treasurer.	Editor of the 'Reports.'
		London.	Provincial.		
1900-1	A. E. Sansom, M.D.	Sydney Stephenson, C.M.	Charles H. Willey, C.M. (Sheffield).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1901-2	A. E. Sansom, M.D.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1902-3	A. H. Tubby, M.S.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1903-4	Sydney Stephenson, C.M.	George Carpenter, M.D. G. A. Sutherland, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1904-5	Sydney Stephenson, C.M.	G. A. Sutherland, M.D. Edmund Cautley, M.D.	R. H. A. Whitelocke, C.M. (Oxford).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1905-6	R. Clement Lucas, B.S.	Edmund Cautley, M.D. Francis Jaffrey.	R. H. A. Whitelocke, C.M. (Oxford).	R. Clement Lucas, B.S.	George Carpenter, M.D.

RULES.

NAME.

1. The name of the Society shall be "The Society for the Study of Disease in Children."

OBJECT OF THE SOCIETY.

2. The object of the Society shall be the study of disease and of allied subjects in children.

CONSTITUTION.

3. The Society shall consist of Ordinary, Temporary, and Honorary Members.

QUALIFICATION FOR MEMBERSHIP.

4. All duly registered medical men shall be eligible for membership.

OFFICERS.

5. The officers of the Society shall be elected from the ordinary members, and shall include an Honorary Treasurer, an Honorary Editor of 'Reports,' Honorary Secretaries (one of whom shall be resident in the London postal district, and one in some other part of Great Britain or Ireland), and twenty-four other members, of whom at least one third shall not be resident in the London postal district. These together shall constitute the Council and manage the affairs of the Society.

ORDINARY MEMBERS.

6. The names of candidates shall be proposed upon a printed form provided for the purpose, and signed by two or more members from personal knowledge. The names of candidates shall in the first instance be submitted to the Council, in which shall be vested the absolute power of declining to allow the

nomination to proceed. The proposal paper, after approval by the Council, shall be read at an Ordinary Meeting of the Society; and the ballot shall take place at the next meeting. No election shall be valid unless at least ten members vote, and no candidate shall be elected who fails to obtain four fifths of the votes recorded. No rejected candidate shall be eligible for proposal until twelve months have elapsed.

FORM OF ADMISSION.

7. Every Ordinary Member, after his election, and after payment of the annual subscription, and of the entrance fee (if any), shall sign the roll of members, declaring thereby his adhesion to the Rules of the Society.

TEMPORARY MEMBERS.

8. Any resident medical officer of a hospital or infirmary shall be eligible for election by the Council as a Temporary Member of the Society for a period of twelve months, without payment of any subscription or entrance fee. He shall be entitled to attend the ordinary Meetings, but he shall not have a vote, shall not be eligible for office, shall not be entitled to receive a copy of the 'Reports,' and shall not introduce visitors. If, at the termination of the period for which he has been elected a Temporary Member, he desires to become an Ordinary Member of the Society, he must go through the usual form of election, but shall be exempt from paying any entrance fee.

HONORARY MEMBERS.

9. The Council shall have power to propose for election as Honorary Members the names of persons eminent in medicine, surgery, or the collateral sciences. The Honorary Members, who shall not exceed twelve in number, shall be balloted for, after proposal by the Council, at a General Meeting of the Society, and be subject to the same rules as govern the election of ordinary members. They shall enjoy all the privileges of ordinary members, voting excepted. They shall not be called upon to pay any fee or subscription.

RESIGNATION AND EXPULSION OF MEMBERS.

10. Any member may retire from the Society on giving written notice of the intention to do so to one of the Secretaries and on paying any contributions that may be due.

The name of any member may be expunged from the Society's roll only at a General Meeting convened by the Council specially for consideration of the case. At least ten votes must be recorded, and four fifths of the votes, if recorded in favour of the expulsion, shall be decisive. Any person expelled in accordance with this rule shall forfeit all claim upon the Society.

SUBSCRIPTIONS.

11. The annual subscription for Ordinary Members shall be One Guinea, payable in advance at the beginning of the session in October. Ordinary Members elected after July the 31st, 1901, shall pay an entrance fee of One Guinea; but in the case of any member elected subsequently to Easter in any year the member shall not be called upon to pay a subscription during the ensuing session. Original Members pay no entrance fee. A member shall at any time be exempted from further annual subscriptions by paying a composition fee of Ten Guineas.

A member whose subscription is six months in arrear shall be reminded of the fact by one of the Honorary Secretaries; and if the amount due remain unpaid during the current year, his name may be removed by the Council from the list of members.

ELECTION OF OFFICERS.

12. The Officers of the Society shall be elected by ballot at the Annual Meeting. No member, except the Honorary Treasurer and the Honorary Editor of the 'Reports,' may hold the same office for a longer period than three consecutive years. A list of names recommended by the Council for election shall be sent to each ordinary member at least fourteen days before the Annual Meeting of the Society. A name or names may be substituted on the voting paper for that of any member or members proposed by the Council for election. Two Scrutineers shall be appointed by the Chairman at the commencement of the Annual Meeting, and their duty shall be to receive and examine the balloting lists, and report the results to the meeting. In the case of equal voting the Chairman shall decide by lot.

HONORARY TREASURER.

13. The Honorary Treasurer shall have charge of the funds of the Society, and keep an account in an orderly and business-like way of all sums that pass through his hands. He shall receive all moneys due to, and pay all moneys due from, the Society, only after the accounts have been passed by the Council and signed by two of the members present. He shall keep a printed receipt-book for subscriptions and fees, and every receipt shall be signed by him and countersigned by one

of the Honorary Secretaries. He shall prepare an annual statement of accounts, which shall be audited by two ordinary members of the Society not in office, and presented, with the Council's Report, to the next Annual Meeting. He shall keep a separate banking account in the name of the Society, and shall invest the funds from time to time as directed by the Council. The Honorary Treasurer shall be *ex officio* a member of Council.

HONORARY EDITOR OF 'REPORTS.'

13A. The Honorary Editor of the 'Reports' shall edit the Society's 'Reports' and he shall keep all papers and communications and illustrations intended for publication in the 'Reports.' He shall be *ex-officio* a member of the Council.

HONORARY SECRETARIES.

14. The Honorary Secretaries (or one of them) shall conduct all correspondence, notify to new members their election, and convene, attend, and minute all meetings of the Society, of the Council, and of any committees appointed by the Council. They shall arrange the order of proceedings at all meetings. They shall submit to the Council all official communications and all nominations for membership in the Society. They shall keep a register of all cases and papers communicated to the Society. They shall keep a roll of the officers and members of the Society. In the discharge of these various duties the Secretaries shall have power to employ clerical assistance, to use printed or typed forms, and to prepay postages and telegrams. They shall be *ex officio* members of Council.

MEETINGS.

15. These shall be *Ordinary*, *Special*, and *General*.

Ordinary Meetings shall be held at hospitals in London or elsewhere, or in places appointed by the Council. There shall not be more than eight Ordinary Meetings in the year, and of these one at least shall be held elsewhere than in London. The meetings shall take place upon the third Friday in the months of October, November, January, February, March, April, May, and June, at 5.30 p.m., unless otherwise arranged by the Council. They shall not be of longer duration than two hours, except by special resolution of the Meeting. Clinical and Pathological Demonstrations, and discussions thereon, shall take precedence over papers. Nothing relating to the laws or the management of the Society shall be discussed at an Ordinary Meeting.

Special Meetings may be convened by the Council for the purpose of discussing a subject or subjects deemed to be of particular interest in relation to disease in children, or to allied subjects.

A *General Meeting* shall be called by the Honorary Secretaries on the requisition of any five members of Council or of any twenty ordinary members of the Society. At least fourteen days' notice shall be given to every ordinary member of the Society; and the character of the business to be transacted at such meeting shall be stated on the summons paper. No business other than that set forth upon the summons paper shall be considered at the meeting.

The *Annual General Meeting* shall be held at a time and in a place determined by the Council, fourteen days' notice having been given to every ordinary member of the Society. Its business shall include election of officers, presentation of the annual Report of the Council, and the discussion of any proposed alteration or alterations in the rules (notice of which shall be given in the summons convening the meeting). Ten ordinary members shall form a quorum. No change in the rules shall be valid unless three fourths of the votes are recorded in favour of the alteration. A notice, written or printed, may be served upon any member, either personally or by sending it through the post in a prepaid letter, addressed to such member at his registered address.

CHAIRMAN.

16. A Chairman, who must be an ordinary member of the Society, shall be chosen by show of hands or by ballot at the commencement of each meeting, whether ordinary, special, or general. He shall sign the minutes of the preceding meeting or meetings, regulate the proceedings, check irregularities, interpret the application of the laws, and enforce the recognised rules of debate. The Chairman shall have an ordinary and a casting vote.

PUBLICATIONS.

17. Reports shall be published in such manner and at such times as the Council may direct. A copy shall be forwarded free to every Honorary Member of the Society, and to every ordinary member whose subscription is not in arrear.

VISITORS.

18. An ordinary member may introduce two medical men as visitors at ordinary meetings of the Society. Visitors shall sign their names in the book provided for the purpose. On the invitation of the Chairman, visitors shall be permitted to take

part in the discussions of the Society. The same visitor shall not be introduced more than twice during one session.

COUNCIL.

19. The Council shall have the management of the affairs of the Society, and shall meet as often as may be necessary for the transaction of business. Seven days' notice of each meeting shall be sent by the Honorary Secretaries to every member of Council. Five members shall form a quorum. The Council shall determine questions, if necessary, by ballot. The Council, at its first meeting, shall elect from amongst its members a Chairman, to preside over its deliberations during the session. He shall not hold office for longer than two consecutive years. In his absence the chair shall be taken by some other member of Council, duly proposed and seconded. The Chairman of Council shall have a casting, in addition to an ordinary vote. The Council shall from time to time make regulations, issue instructions, appoint committees, and take any other lawful steps to further the good government of the Society. It shall prepare and present to the Annual Meeting a Report upon the state of the Society. The Council shall be empowered to fill all vacancies that may occur from death or otherwise between one Annual Meeting and another. It shall decide all questions relating to the publication of the 'Reports.' It shall annually depute three of its members, with the Honorary Secretaries, to form a *Committee of Reference*, the duty of which shall be to determine the acceptance of communications referred to it by the Honorary Secretaries. The Council shall be elected by ballot at the Annual Meeting. One third of its members, exclusive of the Honorary Treasurer, the Honorary Editor of the 'Reports,' and the Honorary Secretaries, shall be replaced annually by an equal number of ordinary members of the Society. An Extraordinary Meeting of Council may be called (1) by the Chairman of Council, or (2) by the Honorary Secretaries, and (3) shall be called by the Honorary Secretaries on the requisition of any five members of Council. Seven days' notice of such extraordinary meeting must be given in writing by the Honorary Secretaries to every member of Council upon a summons paper specifying the object for which the meeting is called.

REPORT OF THE COUNCIL, 1905–6.

Read at the Annual General Meeting, held at No. 11, *Chandos Street, Cavendish Square, W., on Friday, July the* 20*th,* 1906.

The Council have pleasure in reporting that the prosperity of the Society is well maintained. The Society has to mourn the loss by death of four members, namely: Dr. J. H. Bryant, the accomplished Assistant Physician of Guy's Hospital; Dr. C. N. Gwynne, of Sheffield, a Member of the Council and the admirable Chairman of the Provincial Meeting held at Sheffield last year; Dr. T. J. Barnardo, the widely-known philanthropist; and Mr. G. L. Rugg.

The Meetings of the Society have been well attended, and many interesting cases, specimens, and papers have been brought before the Society and discussed. A Special Meeting was held in December, for a Discussion on "Pleural Effusions, Serous and Purulent" in all aspects. The Wightman Lecture was delivered by M. le Docteur Broca, Surgeon to the Hôpital des Enfantes Malades, and President of the Société de Pédiatrie of Paris, on the subject of "Appendicitis." M. Broca was subsequently entertained at a Dinner, at which about one hundred Members and guests were present.

The Provincial Meeting was held at the Radcliffe Infirmary, Oxford, on Saturday, June the 23rd. Several interesting cases and specimens were demonstrated and discussed, and subsequently the meeting proceeded to the reading and discussion of the papers. The chair was taken by Dr. William Osler, the Regius Professor of Medicine. The Meeting was a very pleasant one, and the Council desire to express their indebtedness to Mr. R. H. A. Whitelocke, who undertook the local arrangements, and to those members of the profession in Oxford who took part in providing the programme. A large number of local medical men were present as guests of the Society.

The finances of the Society are in a very satisfactory condition.

REPORT ON THE AMALGAMATION SCHEME.

The proposal to amalgamate the chief Medical Societies of London, general and special, into one large Society, to be called the Royal Society (or Academy) of Medicine, has received the most careful consideration of the Council at numerous meetings.

From the outset the Council held the opinion that it was of little practical value to enter into active negotiations until it was ascertained that the two richest societies, the Medical and the Royal Medical and Chirurgical, had agreed on a basis of amalgamation. So far no such agreement has been arrived at.

In considering the scheme, the Council have paid special attention to the following points:

(1) Is such an amalgamation in the best interests of the medical profession?

(2) Is the proposed scheme of amalgamation financially sound?

(3) Would amalgamation be beneficial to this Society, and could its privileges be efficiently safeguarded?

The Council are by no means unanimous that amalgamation is in the best interests of the profession, or that the financial basis of the scheme is satisfactory. Further, it does not appear that any provision has been made to safeguard the position of the Society as a permanently independent section, and the continuance of its annual publication of a volume of reports.

In order to ascertain the views of the Members, a post-card circular was sent out inviting an opinion on the question. In this circular the reasons which influenced the Council in forming their opinion were not formulated.

The result showed that a preponderance of Country Members were opposed to amalgamation, viz. 43 against 34, and a preponderance of London Members were in favour of it, viz. 76 against 47.

Of the existing 28 Members of Council, 18 were opposed to amalgamation, 4 were in favour of it, and 6 did not record any opinion.

TREASURER'S REPORT.

The Treasurer has much pleasure in reporting the continued and progressive financial prosperity of the Society.

A balance of £206 4*s*. 2*d*. was carried forward from last year. Of this £143 5*s*. 9*d*. were invested in Consols, bringing up the amount invested to exactly £500 Consols. A collector, Mr. Harris, was employed for the first time this year to collect arrears of subscriptions, with the result that the subscriptions and entrance fees amount to £365 3*s*. this year, as compared with £347 11*s*. last year. There has been also an increase in the sale of the 'Reports,' £8 19*s*. having been received from this source for the year as compared with £7 8*s*. 11*d*. received for the two combined years 1904 and 1905. The total income from all sources together with the balance amounted to £594 3*s*. 10*d*. Of this £372 2*s*. 3*d*. have been expended, leaving a balance of £222 1*s*. 7*d*. to be carried forward.

Of the Wightman Lecture Fund the balance of £20 was paid to M. le Docteur Broca, and a further subscription of £20 has been received from Mr. Wightman for the year 1907.

R. CLEMENT LUCAS.

BALANCE SHEET FOR THE YEAR ENDING JULY, 1906.

RECEIPTS.	£	s.	d.	PAYMENTS.	£	s.	d.
To Balance brought forward	206	4	2	By Hollebone Bros. & Trench, purchase of £158 9*s.* 10*d.* 2½ % Consols, making £500 invested	143	5	9
,, Subscriptions and Entrance Fees ...	365	3	0	,, G. Pulman and Sons	25	4	7
,, Dinner Fund Surplus	2	0	0	,, Adlard and Son	155	8	8
,, Dividend on Consols	11	17	8	,, Wm. Whiteley, Limited	0	14	6
,, Messrs. Churchill, Sale of Reports ...	8	19	0	,, H. Dickinson	19	3	3
				,, Dr. Cautley	2	16	6
				,, The Medical Society	18	18	0
				,, W. Harris (Collector)	3	11	0
				,, Treasurer's Petty Cash	3	0	0
					372	2	3
				,, Balance carried forward	222	1	7
	£594	3	10		£594	3	10

INVESTMENT.

Consolidated Stock, 2½ % £500

WIGHTMAN LECTURE FUND.

	£	s.	d.		£	s.	d.
Balance brought forward	£20	0	0	Paid Dr. Broca	£20	0	0
Per Mr. Wightman	20	0	0	Balance in hand	20	0	0
	£40	0	0		£40	0	0

We, the undersigned, have examined the foregoing accounts, together with the Vouchers and Bank Book, and find them to be correct.

JOHN POLAND, } *Auditors.*
H. S. CLOGG, }

July the 17*th*, 1906.

Meetings of the Society were held during the Session of 1905–1906 *at the undermentioned places.*

1. Friday, October the 20th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Dr. Percy Lewis (Folkestone) in the Chair.
2. Friday, November the 18th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.
3. Friday, December the 15th, 1905 (Special Meeting for the discussion of "Pleural Effusions, Serous and Purulent").—No. 11, Chandos Street, Cavendish Square, W. Dr. Leonard Guthrie in the Chair.
4. Friday, January the 19th, 1906.—No. 11, Chandos Street, Cavendish Square, W. Dr. Porter Parkinson in the Chair.
5. Friday, February the 16th, 1906.—No. 11, Chandos Street, Cavendish Square, W. Mr. A. H. Tubby in the Chair.
6. Friday, March the 16th, 1906.—No. 11, Chandos Street, Cavendish Square, W. Dr. G. A. Sutherland in the Chair.
7. Friday, April the 20th, 1906.—No. 11, Chandos Street, Cavendish Square, W. Dr. Porter Parkinson in the Chair.
8. Friday, May the 4th, 1906 (a Meeting devoted to the Exhibition of Pathological Specimens).—No. 11, Chandos Street, Cavendish Square, W. Mr. George Pernet in the Chair.
9. Friday, May the 18th, 1906 (the Wightman Lecture "On Chronic Appendicitis and the Early Diagnosis and Treatment of Acute Appendicitis in Children," by Dr. A. Broca, Paris).—No. 11, Chandos Street, Cavendish Square. Mr. R. Clement Lucas in the Chair.
10. Saturday, June the 23rd, 1906 (Provincial Meeting).—The Radcliffe Infirmary, Oxford. Professor W. Osler in the Chair during the exhibition of clinical cases and pathological specimens, and Dr. W. Collier in the Chair during the reading of papers.
11. Friday, July the 20th, 1906 (Annual General Meeting).—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.

LIST OF OFFICE BEARERS, PAST MEMBERS OF COUNCIL, WITH DATES OF SERVICE, AND MEMBERS OF THE SOCIETY FOR THE SESSION 1906—1907.

(*Revised October*, 1906.)

(O.M. = Original Member. T. = Treasurer. S. = Secretary. S with date attached = Past Secretary. P.S. = Provincial Secretary. P.S. with date attached = Past Provincial Secretary. Ed. = Editor of the Reports. C. = Member of the Council. C. with date attached = Past Member of Council. * = Life Member. C.C. = Chairman of Council. C.C. with date attached = Past Chairman of Council. W. = Wightman Lecturer. W. with date attached = Past Wightman Lecturer.

Elected

O.M. Abrahams, Bertram Louis, M.B., Assistant-Physician at Westminster Hospital; 14, Welbeck Street, W.

O.M. Achard, Alexander Louis, M.D., 34a, Gloucester Place, W.

O.M. Alliott, Alexander John, M.D., Surgeon at The Hip Hospital, Sevenoaks; The Vine, Sevenoaks. (C.)

1904 Anderson, C. M., M.D., 146, Gloucester Terrace, Hyde Park, W.

1903 Anderson, J. Ford, M.D., 41, Belsize Park, N.W. (C. 1903–4, 1904–5.)

1901 Archdale, Mervyn A., M.B., East Riding Lunatic Asylum, Beverley.

1902 Armour, Donald John, M.B., Assistant-Surgeon at The West-London Hospital; Surgeon at The Belgrave Hospital for Children; 89, Harley Street, W.

O.M. Armstrong, Henry George, Medical-Officer at Wellington College; Wellington College, Berks. (C. 1900–1, 1901–2.)

Elected

1901 ARMSTRONG, JOHN T., Selby House, Beaumont Terrace, West Kensington, W.

O.M. ASHBY, HENRY, M.D., Physician at The Manchester General Hospital for Children; Lecturer on Diseases of Children at Owens College; 13, St. John Street Manchester. (W. 1904–5.) (C. 1900–1, 1901–2, 1902–3.)

1900 BARR, SIR JAMES, M.D., Physician at The Royal Infirmary, Liverpool; 72, Rodney Street, Liverpool.

1902 BARRETT, ALFRED KEPPEL, 5, Bedford Gardens, Kensington, W.

1904 BATCHELOR, CHARLES, 1, Fairfield Avenue, Staines, Middlesex.

1901 BATTAMS, J. SCOTT, 32, Cedars Road, Clapham Common, S.W.

1904 BAUMANN, E. P., M.D., Box 1257, Johannesburg, Transvaal, S. Africa.

O.M. BEACH, FLETCHER, M.B., Physician at The West-End Hospital for Diseases of the Nervous System; Winchester House, Kingston Hill, Surrey. (C. 1900–1, 1901–2, 1902–3.)

1906 BEATTY, ROBERT MOORHEAD, M.B., Assistant-Physician at The Belfast Hospital for Sick Children; 9, Clifton Street, Belfast.

1901 BEDDARD, ARTHUR PHILIP, M.D., Senior Assistant-Physician and Pathologist at The West-London Hospital; 44, Seymour Street, W.

1904 BIDWELL, LEONARD, Surgeon at The West London Hospital; 15, Upper Wimpole Street, W.

1900 BLAGG, ARTHUR F., M.D., Physician at The Orthopædic Hospital for Children, Bristol; 28, Caledonia Place, Clifton, Bristol.

1902 BLAIKIE, JAMES BRUNTON, M.B., Physician at The Hospital for Consumption, Margaret Street, W.; 80, Brook Street, W.

O.M. BLUCK, WALTER CARDY, M.B., Byford House, York Road, Southend-on-Sea.

O.M. BLUMFELD, JOSEPH, M.D., Senior Anæsthetist at St. George's Hospital; 7, Cavendish Place, W. (C. 1905–6.) (C.)

Elected

1903 Braithwaite, John, M.D., Physician at The Devonshire Hospital and Buxton Bath Charity, 2, Hardwick Mount, Buxton.

1906 Brewerton, Elmore Wright, Ophthalmic Surgeon at Metropolitan Hospital; Assistant Surgeon Royal Westminster Ophthalmic Hospital; 84, Wimpole Street, W.

O.M. Brightman, Frank, Aspley House, Broadstairs, Kent.

1904 Briscoe, J. Charlton, M.B., Physician at The Evelina Hospital for Sick Children; 110, Harley Street, W.

1901 Brown, John Henry, M.D., 14, Burngreave Road, Sheffield.

O.M. Browne, Hablot J. M., M.B., Hoylake, Cheshire.

1906 Brunton, Sir Lauder, M.D., Consulting Physician at St. Bartholomew's Hospital; 10, Stratford Place, W.

1904 Burgess, Harold, L., c/o Messrs. Worsfold and Hayward, Market Square, Dover, Kent.

1904 Burges, Richard, 4, Worcester Terrace, Clifton.

1901 *Burgess, William Milner, 43, Nicoll Road, Harlesden, N.W. (C. 1904–5.)

O.M. Burghard, Fred. François, M.S., Surgeon at King's College Hospital and at Paddington-Green Children's Hospital; 86, Harley Street, W.

1905 Burnet, James, M.D., Registrar, Royal Hospital for Sick Children, Edinburgh; 6, Glengyle Terrace, Edinburgh.

1904 Burrows, Harold, M.B., Assistant-Surgeon at The Bolingbroke Hospital; 32, Wimpole Street, W.

1901 Burt, Albert Hamilton, 52, Lansdowne Place, Hove, Sussex.

O.M. Burton, Charles George, 77, Church Street, Lower Edmonton, N.

1902 Buzzard, E. Farquhar, M.B., Assistant-Physician at The Belgrave Hospital for Children, Assistant-Physician at The Royal Free Hospital; 33, Harley Street, W.

1902 Bythell, W. J. S., "Woodcliffe," Prestwich, Manchester.

1905 Campbell, Harry, M.D., Physician at The North-West London Hospital, and at The West End Hospital for Nervous Diseases; 23, Wimpole Street, W.

Elected

1902 Campbell, Henry Johnstone, M.D., Senior Physician at The Bradford Royal Infirmary.

1901 Campbell, Robert, M.B., Senior Surgeon at The Belfast Hospital for Sick Children: 21, Great Victoria Street, Belfast. (C. 1904–5, 1905–6.)

O.M. Carmichael, James, M.D., Consulting-Physician at The Royal Edinburgh Hospital for Sick Children: 22, Northumberland Street, Edinburgh. (C. 1901–2.)

O.M. Carpenter, George, M.D, Membre Correspondant de la Société de Pédiatrie de Paris, Physician at The North-Eastern Hospital for Children; late Physician at The Evelina Hospital for Sick Children; Editor and Founder of 'The British Journal of Children's Diseases'; 12, Welbeck Street, W. (C. 1900–1.) (S. 1901–2, 1902–3, 1903–4.) (Ed. 'Reports' 1900–1, 1901–2, 1902–3, 1903–4, 1904–5, 1905–6.) (Ed.)

1906 Carpmael, Norman, B.S., 375, Norwood Road, West Norwood, S.E.

O.M. Carre-Smith, Herbert L., 74, Holland Park Avenue, Holland Park, W. (C. 1902–3, 1903–4, 1904–5.)

1900 Cathcart, George C., M.B., Senior Assistant-Surgeon at The London Throat Hospital, 35, Harley Street, W.

O.M. *Cautley, Edmund, M.D., Physician at The Belgrave Hospital for Children: Physician at The Metropolitan Hospital; 15, Upper Brook Street, W. (C. 1900–1, 1901–2, 1902–3.) (S. 1904–5, 1905–6.) (C.)

1901 Cave, Edward John, M.D., Physician at The Royal United Hospital, Bath; 20, Circus, Bath.

O.M. Chaffey, Wayland C., M.D., Physician at The Royal Alexandra Hospital for Children, Brighton; 18, Palmeira Square, Hove, Sussex. (C. 1900–1, 1901–2, 1902–3.)

1901 Chaldecott, John Henry, Anæsthetist at St. John's Hospital for Diseases of the Skin, at The Metropolitan Throat, Nose, and Ear Hospital, and at The Italian Hospital; 60, Harley Street, W.

O.M. Chapman, Charles William, M.D., Physician at The Hospital for Diseases of the Heart, Soho Square; 21, Weymouth Street, W. (C. 1903–4, 1904–5, 1905–6.)

Elected

1901 Cheyne, William Watson, C.M., Surgeon at King's College Hospital, and at Paddington-Green Children's Hospital; 75, Harley Street, W. (C. 1901–2.)

1901 Clapham, E. W., 48, Harley Street, W.

O.M. Clarke, Ernest, M.D., Surgeon at The Central London Ophthalmic Hospital; Ophthalmic-Surgeon at The Miller Hospital, Greenwich; 3, Chandos Street, Cavendish Square, W.

1904 Clogg, H. S., M.S., Assistant-Surgeon at Charing Cross Hospital, and Surgeon at the Evelina Hospital for Sick Children; 117a, Harley Street, W.

1905 Clutterbuck, L. A., M.D., Physician at the Marylebone General Dispensary; Welbeck Street, W.

1902 Cockburn, Robert P., 64, The Avenue, Ealing, W.

1902 Collier, H. Stansfield, Surgeon to Out-patients at St. Mary's Hospital, and Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 57a, Wimpole Street, W.

1901 Collier, Mark Purcell Mayo, M.S., Senior Assistant-Surgeon at The North-West London Hospital; Consulting-Surgeon at The National Hospital for Diseases of the Nervous System; 133, Harley Street, W.

1906 Collier, William, M.D., Physician to Radcliffe Infirmary; St. Mary's Entry, Oxford.

1901 Cooper, Percy Robert, M.D, "Glenthorne," The Downs, Bowdon, Cheshire.

1901 Corner, Harry, M.D., Brook House, Southgate, N.

1905 Cowper, W. P., 30, Frognal Lane, Hampstead, N.W.

1901 Cox, Joshua J., M.D., Physician for Diseases of Children at The Clinical Hospital, Manchester; 38, Deansgate, Manchester.

1904 Critchley, Harry George, M.D., 10, Bentinck Street, W.

1902 Cross, Ernest, W., "The Limes," Wallwood Park, Leytonstone.

1901 Crossley, Leonard, M.B., "Beechwood," Arnside, Carnforth, Lancashire.

1902 Crowley, Ralph H., M.D., Physician at The Bradford Royal Infirmary; 116, Manningham Lane, Bradford.

Elected

1905 Cuff, Archibald, Surgeon Royal Infirmary, and Assistant-Surgeon Children's Hospital; 285, Glossop Road, Sheffield.

1904 Curl, Sydney Walter, M.D., 58, Crouch Street, Colchester.

1902 Curtis, Henry J., B.S , late Surgeon at The Royal Hospital for Women and Children; late Surgeon at The North-Eastern Hospital for Children ; 58, Harley Street, W.

1901 Dawnay, Archibald Hugh Payan, Anæsthetist at The Royal Orthopædic Hospital ; 126, Harley Street, W.

O.M. Deas, Frank, Thorndale, Merton Park, S.W. (C. 1905–6.) (C.)

O.M. Dent, Clinton T., M.C., Surgeon at St. George's Hospital and Consulting-Surgeon at The Belgrave Hospital for Children; 61, Brook Street, W. (C. 1902–3, 1903–4, 1904–5.)

O.M. Dockrell, Morgan, M.D., Physician at St. John's Hospital for Diseases of the Skin ; 9, Cavendish Square, W.

1901 Dodd, Arthur Herbert, 49, Church Road, Hove. Sussex.

1901 Douglas, Arch. R., Assistant-Medical Officer and Deputy Superintendent at The Royal Albert Asylum, Lancaster.

1901 Drake-Brockman, E. F., Lieutenant-Colonel I.M.S., late Professor of Physiology and Diseases of the Eye at The Medical College, Madras, and late Superintendent at The Eye Infirmary, Madras, India ; 14, Welbeck Street, W.

O.M. Drew, Douglas, B.S., Surgeon at The North-Eastern Hospital for Children and at The Hospital for Women, Soho Square ; 1, Harley Street, W.

1905 Dudgeon, Leonard S., Superintendent of Clinical Laboratory, St. Thomas's Hospital ; 6, Powis Gardens, Bayswater, W.

1901 Dun, Robert Craig, M.B., Surgeon at The Liverpool Infirmary for Children ; 6, Rodney Street, Liverpool.

1901 Dunlop, James, Aveley, Purfleet, Essex.

1901 Dunn, W. J. Robertson, 46, Wellington Terrace, West Derby Road, Liverpool.

1906 East, C. H., M.D., St. Clare, Malvern.

1906 Eccles, W. McAdam, M.S., 124, Harley Street.

Elected

1901 Edelsten, Ernest Alfred, M.B., 370, Brixton Road, S.W.

1904 Edmunds, Arthur, B.S., Assistant-Surgeon at The Paddington Green Children's Hospital; 23, Upper Wimpole Street, W.

O.M. Edmunds, Walter, M.C., Surgeon at The Tottenham Hospital; 2, Devonshire Place, W. (C. 1900–1, 1901–2, 1902–3.)

1905 Elliott, Andrew, M.D., Medical Registrar, West London Hospital; 11, Upper Berkeley Street, Portman Square, W.

1906 Elliott, Frank Percy, M.B., Medical Officer Walthamstow Public Dispensary; 113, Grove Road, Walthamstow.

1901 Ellis, W. McDonogh, M.D., Assistant-Physician at The Hospital for Women, Bath; Cedar Lodge, Bath.

1904 Emanuel, J. G., M.D., Physician to Out-patients at Queen's Hospital, Birmingham; 47, Newhall Street, Birmingham.

1905 English, T. Crisp, B.S., Assistant-Surgeon at St. George's Hospital, and at the Grosvenor Hospital for Women and Children; 31, Grosvenor Street, W.

1901 Evans, J. Howell, M.Ch., Assistant-Surgeon at The Cancer Hospital; 63, Grosvenor Street, W.

1901 Evans, Thomas Hy., 95, Shaw Street, Liverpool.

1904 Evelyn, William Arthur, M.D., 24, Micklegate, York.

O.M. Ewart, William, M.D., Senior Physician at St. George's Hospital and at The Belgrave Hospital for Children; 33, Curzon Street, Mayfair, W. (C. 1904–5, 1905–6.) (C.)

1901 Fisher, H. Wycliffe, "Rathescar," Greenbank Drive, Sefton Park, Liverpool.

O.M. Fisher, Theodore, M.D., late Pathologist at The Bristol Royal Infirmary, and late Physician to Out-patients at The Bristol Hospital for Sick Children; Assistant-Physician at The East London Hospital for Children; The Garples, Sidcup, Kent. (C. 1900–1.) (P.S. 1901–2, 1902–3, 1903–4.) (C. 1904–5.)

1906 Fitzwilliams, Duncan C. L., M.B., 17, Queen Street, Mayfair.

Elected

1906 Fordyce, A. Dingwall, M.B., Physician for Diseases of Children at Newtown Dispensary; 19, Coates Crescent, Edinburgh.

1901 Forster, Henry John, 119, Victoria Road, Romford, Essex.

1903 Fortescue-Brickdale, John Matthew, M.D., Registrar and Pathologist at The Royal Hospital for Children and Women, Bristol; 52, Pembroke Road, Clifton, Bristol.

O.M. Fox, T. Colcott, M.B., Physician for Diseases of the Skin at Westminster Hospital, and at Paddington-Green Hospital for Children; 14, Harley Street, W.

1905 Fox, W. S., 63, Grosvenor Street.

1901 *Frost, Edmund, M.D., "Chesterfield," Chesterfield Road, Eastbourne.

O.M. Fuller, Andrew, Local Government Board Inspector; "The Bushes," Chase Road, Oatlands Park, Weybridge, Surrey.

1906 Furber, Edward Price, Beechcroft, Oxted, Surrey.

1901 George, Alfred Walter, M.D., Surrey House, Shoot-up Hill, N.W.

1905 Gerrard, Alfred H., M.B., Surgeon at the South Wimbledon and Merton Cottage Hospital; 90, Merton Road, Wimbledon, S.W.

1904 Gerrish, Daniel S., Coningsby Villa, 332, Church Road, St. George, Bristol.

1900 Gervis, F. Harcourt, M.D., 1, Lyncroft Mansions, West Hampstead, N.W.

1906 Gibson, A. G., M.B., 28, Banbury Road, Oxford.

1905 Gibson, George A., M.D., Physician at the Royal Infirmary, Edinburgh; 3, Drumsheugh Gardens, Edinburgh. (C. 1905–6.)

O.M. Giffard, Henry Edward, Denham House, Egham.

O.M. Giles, Leonard Thomason, 5, St. Nicholas Parade, Scarborough.

1905 Goldsmith, G. Harvey, M.D., Ophthalmic Surgeon, at Bedford County Hospital; Harpur Place, Bedford.

1906 Goodhart, James Frederick, M.D., Consulting Physician at Guy's Hospital and at The Evelina Hospital for Children; 25, Portland Place, W.

Elected

1901 Grant, J. Dundas, C.M., Surgeon at The Central London Throat and Ear Hospital; 18, Cavendish Square, W.

O.M. Gray, Alexander C. E., M.D., 1, Elm Park Gardens, South Kensington, W. (C.)

1904 Grünbaum, Otto F. F., M.D., Assistant-Physician at The Belgrave Hospital for Children; Medical Registrar at The London Hospital; 34a, Wimpole Street, W.

1901 Gullan, Archibald Gordon, M.D., 5, Canning Street, Liverpool.

O.M. Guthrie, Leonard George, M.D., Physician at The Hospital for Epilepsy and Paralysis, Regent's Park; Senior Physician at The Paddington-Green Children's Hospital; 15, Upper Berkeley Street, W. (C. 1900–1, 1901–2, 1902–3.) (C. 1904–5, 1905–6.) (C.)

1904 Harman, N. Bishop, M.B., Ophthalmic Surgeon, Belgrave Hospital for Children; 108, Harley Street, W.

O.M. Hawken, Giles Lockwood Lang, Surgeon at St. John's College, Hurstpierpoint, Sussex.

1901 Hawthorne, Charles Oliver, M.D., Physician at The Central London Ophthalmic Hospital; Assistant-Physician at The Royal Hospital for Children and Women; 63, Harley Street, W. (C. 1905–6.) (C.)

1904 Haynes, G. Secretan, 4, Trinity Street, Cambridge.

1906 Helt, G. Seccombe, M.B., 16, Palace Gardens Mansions, The Mall, Kensington, W.

1901 Heywood, Charles Christopher, M.B., Physician at Manchester Children's Hospital and Assistant-Physician for Diseases of Children at Manchester Northern Hospital; Winter's Buildings, St. Ann Street, Manchester.

1904 Hill, G. Wm., M.D., Surgeon for Diseases of the Ear at St. Mary's Hospital; 26, Weymouth Street, Portland Place, W.

1902 Hinds, Frank, M.D., Honorary-Surgeon at The Infirmary, Worthing; 17, Tennyson Road, Worthing. (C. 1904–5, 1905–6.)

1901 Hobhouse, Edmund, M.D., Assistant-Physician at The Sussex County Hospital: Physician at The Royal Alexandra Hospital for Children, Brighton; 12, Second Avenue, Hove. (C. 1903–4, 1905–6.) (C.)

Elected

1904 Hobson, H. Overton, M.D., 32, Upper Berkeley Street, W.

1905 Hollings, Guy B., M.D., 22, Endsleigh Gardens, N.W.

1901 Hopkinson, Albert, M.B., 2, Parsonage Road, Withington, Manchester.

1904 Howard, John Alexander, M.D., 40, Harold Road, Upper Norwood.

1905 Howard, J. Russell, M.S., Assistant-Surgeon at The London Hospital and The Royal Waterloo Hospital for Women and Children; 10, Weymouth Street, W.

1901 Hutchison, Robert, M.D., Assistant-Physician at The London Hospital, and at The Hospital for Sick Children, Great Ormond Street; 22, Queen Anne Street, W. (C. 1901–2, 1902–3, 1903–4.)

O.M. Hutton, David Mount, M.D., 5, Lulworth Road, Birkdale.

O.M. Hutton, Henry Richmond, M.B., Physician at The Manchester General Hospital for Children; 16, St. John Street, Manchester. (C. 1903-4, 1904–5, 1905–6.)

1904 imThurm, Robert McKenzie, Ailsa, Sydenham.

O.M. Jaffrey, Francis, Surgeon at St. George's Hospital; Surgeon at The Belgrave Hospital for Children; 33, Nottingham Place, W. (C. 1901–2, 1902–3, 1903–4.) (S. 1905–6.) (S.)

1901 James, Charles Alfred, 49, Stamford Hill, N.

1905 Jessop, Edward, 81, Fitzjohn's Avenue, Hampstead, N.W.

O.M. Jessop, Walter Hamilton Hylton, M.B., Ophthalmic-Surgeon at St. Bartholomew's Hospital; 73, Harley Street, W.

1901 Johnson, L. Capper, Lidgate Park Road, Roundhay, Leeds.

1902 Johnson, Raymond, Surgeon at University College Hospital and at The Victoria Hospital for Children; 11, Wimpole Street, W.

1904 Jones, A. Ernest, M.D., 13, Harley Street, W.

O.M. Jones, Robert, Surgeon at The Royal Southern Hospital, Liverpool; 11, Nelson Street, Liverpool. (C. 1900–1, 1901–2.)

Elected

1903 Jones, W. Llewellyn, M.D., 58, Thomas Street, Merthyr Tydfil, South Wales.

1905 Kellie, Kenneth H. A., M.B., Westfield, Curzon Park, Chester.

1902 Kelson, William Hy., M.D., Assistant-Surgeon at The London Throat Hospital; 17, Cavendish Place, Cavendish Square, W.

O.M. Knapton, George, Yew Bank, Bowdon, Cheshire.

1904 Knight, Frederick, M.D., Physician at The Swansea Hospital; 10, Walter Terrace, Swansea.

O.M. Lack, Harry Lambert, M.D., Surgeon to the Throat Department at The London Hospital; Surgeon at The Throat Hospital, Golden Square; 48, Harley Street, W.

1901 Lacy, Alexander Gairdner, "Saumarez," Sunninghill, Ascot, Berks.

1901 Lake, Richard, Surgeon at The Royal Ear Hospital; 60, Harley Street, W.

O.M. Lambert, Sydney H. A., M.B., Gothic House, Harrow-on-the-Hill. (C.)

1901 Langdon-Down, Percival L., M.B., 47, Welbeck Street, W.

1900 Langdon-Down, Reginald Langdon, M.B., 47, Welbeck Street, W.

1905 Langmead, F., M.B., Hospital for Sick Children, Great Ormond Street.

O.M. Lawson, Arnold, M.D., Assistant-Surgeon at The Royal London Ophthalmic Hospital, and Ophthalmic-Surgeon at The Children's Hospital, Paddington-Green; 12, Harley Street, W.

1905 Lee, H. E., M.B., 45, Carlton Hill, N.W.

O.M. Lee, Philip George, Surgeon at The Women and Children's Hospital, Cork; 25, St. Patrick's Hill, Cork. (C.)

1904 Leedham-Green, Charles A., M.D., Surgeon at Queen's Hospital, Birmingham; 9, Newhall Street, Birmingham.

1901 Leslie, W. Murray, M.D., 74, Cadogan Place, Belgrave Square, S.W.

1904 Lett, Hugh, Ch.B., Assistant-Surgeon at The Belgrave Hospital; 25, Queen Anne Street, W. (C.)

Elected

O.M. Lewis, Percy G., M.D., Honorary Medical Officer at The Victoria Hospital, Folkestone; 22, Manor Road, Folkestone. (C. 1903–4, 1904–5, 1905–6.)

1904 Lingard, Alfred, M.B., Imperial Bacteriologist to the Indian Government, Imperial Bacteriological Laboratory, Nainital District, United Provinces, India.

O.M. Lister, Thomas David, M.D., Assistant-Physician at The Royal Hospital for Women and Children, Assistant-Physician at The Mount Vernon Hospital; 50, Brook Street, W.

1901 Livingstone, George R., 35, George Street, Dumfries, N.B.

1905 Low, V. Warren, B.S., Assistant-Surgeon at St. Mary's Hospital; 27, Queen Anne Street, Cavendish Square, W.

O.M. *Lucas, R. Clement, B.S., Senior Surgeon and Lecturer on Surgery at Guy's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 50, Wimpole Street, W. (T.) (W. 1903–4.) (C.C. 1905–6.)

1905 Maddox, R. H., M.B., Major I.M.S., Ranchi, Chota Nagpur, India; c/o Messrs. Thos. Cook & Sons, Ludgate Circus.

O.M. McCaw, John, M.D., Physician at The Belfast Hospital for Children; 74, Dublin Road, Belfast. (C. 1901–2.)

1904 McGregor, John, M.D., 15a, Pembridge Crescent, W.

1905 McIlraith, C. H., M.D., 44, Half Moon Street, Herne Hill, S.E.

O.M. McKenzie, Dan, M.D., Holly Lodge, Leytonstone.

O.M. McKerron, Robert Gordon, M.D., Senior Physician at The Children's Hospital, Aberdeen; 1, Albyn Place, Aberdeen. (C. 1902–3.) (C.)

1901 McNab, Alexander Anderson, M.B., 15, Wesley Road, Armley, Leeds.

1900 Macalister, Charles John, M.D., Physician at The Liverpool Royal Southern Hospital; 35, Rodney Street, Liverpool. (C. 1901–2, 1902–3, 1903–4.)

1901 Mackintosh, John Stewart, St. Ive, Platt's Lane, Hampstead, N.W.

1901 Mackintosh, William Alexander, M.B., 3, Park Terrace, Stirling. (C. 1904–5.)

1901 Maitland, Alfred Derwent, 1, Gloucester Villas, Hanwell, W.

Elected

1904 Makin, Frank Humphrey, M.B., c/o 'South African Register,' 80, Fleet Street, E.C.

1901 Makins, George Henry, C.B., Surgeon at St. Thomas's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 47, Charles Street, Berkeley Square, W.

O.M. Malcolm, John D., C.M., Surgeon at The Samaritan Free Hospital; 13, Portman Street, Portman Square, W.

1904 Marsh, J. J., 104, Sloane Street, S.W.

O.M. Marshall, Lewis W., M.D., Surgeon at The Children's Hospital, Nottingham; 42, The Ropewalk, Nottingham. (C. 1900–1, 1901–2, 1902–3.)

1900 May, W. Page, M.D., 9, Manchester Square, W.

1902 Maynard, J. Seymour, M.B., 4, Charles Street, St. James's Square, S.W.

1904 Midelton, William John, 72, Charminster Road, Bournemouth.

O.M. Milburn, Charles Henry, M.S., Surgeon at The Victoria Hospital for Children, Hull; 158, Anlaby Road, Hull.

1902 Miller, Charles Hewitt, Oughtrington House, Lymm, Cheshire.

1902 Milligan, Wyndham Anstruther, M.D., Registrar at The Hospital for Women, Soho Square; Pathologist at The North-Eastern Hospital for Children; 68, Park Street, Grosvener Square, W.

1901 Milne, Robert, M.D., 32, Bow Road, E.

O.M. Moline, Paul F., M.B., Anæsthetist at The Victoria Hospital for Children, Chelsea; 42, Walton Street, Chelsea, S.W.

1901 Monsarrat, Keith Waldegrave, C.M., Surgeon at The Northern Hospital, Liverpool; 11, Rodney Street, Liverpool. (C. 1904–5.)

1901 Montgomery, W. P., B.S., Surgeon at The Children's Hospital, Manchester, Assistant-Surgeon at The Manchester Royal Infirmary; 15, St. John Street, Manchester.

1906 Moon, Edward Gibson, High Beach, Victoria Parade, Broadstairs.

1901 Morgan, Cecil Augustus, M.B., 6, Somerset Street, Portman Square, W.

Elected	
1902	Morgan, George, Senior Surgeon at The Children's Hospital, Brighton; 6, Pavilion Parade, Brighton.
1901	Morison, Alexander, M.D., Physician to Out-patients at The Great Northern Central Hospital, and at The Children's Hospital, Paddington-Green; 14, Upper Berkeley Street, W. (C. 1903–4, 1904–5, 1905–6.)
1904	Morton, C. A., Surgeon at The Bristol General Hospital, and at The Bristol Royal Hospital for Sick Children; 14, Vyvyan Terrace, Clifton.
O.M.	Mossop, Isaac, Surgeon at The Bradford Children's Hospital; Victor Lodge, Manningham, Bradford. (C.)
1905	Mounsey, R. E., Moorlands, Camberley, Surrey.
1902	Mummery, John Percy Lockhart, B.C., 10, Cavendish Place, W., Assistant-Surgeon at The North-Eastern Hospital for Children. (C. 1904–5, 1905–6.) (C.)
1901	Munro, Henry Acland, M.B., "Lulworth," Rushey Green, Catford, S.E.
1904	Murphy, J. Keogh, B.C., Surgeon to Out-patients at The Paddington-Green Children's Hospital; 9, Weymouth Street, Portland Place, W.
1901	Murtz, Anton Peter, Clareville, 48, Queen's Road, Finsbury Park, N.
1901	Nabarro, David N., M.D., Assistant-Professor of Pathology and Lecturer on Bacteriology, University College; 6, Crown Office Row, Inner Temple, E.C.
1904	Nash, Elwin H. T., Oulton House, Burnley Road, Accrington, Lancs.
1901	Nash, W. Gifford, Surgeon at The Bedford County Hospital; Clavering House, Bedford.
1905	Neave, Sheffield, Assistant-Physician at The North-Eastern Hospital for Children; Millgreen Park, Ingatestone, Essex.
1902	Newbolt, George P., Surgeon at The Royal Southern Hospital, Liverpool; 42, Catherine Street, Liverpool.
1901	Nisbet, J. Tawse, M.D., 7, Croxteth Road, Liverpool.
1901	Niven, Robert, Camberwell Infirmary, Brunswick Square, S.E.
1906	Nix, Edward J., M.D., Physician at All Saints' Children's Hospital; 11, Weymouth Street, W.

Elected

1906 Odgers, P. N. Blake, M.B., B.Ch.Oxon., Assistant Surgeon Northampton Royal Hospital; 16, Castilian Road, Northampton.

1901 Oldmeadow, Lloyd J. H., M.D., Kineton, Warwickshire.

1901 Oppenheimer, Heinrich, M.D., 63, Finsbury Pavement, E.C.

1904 Orme, Gilbert Edward, B.C., Lowfield, Clitheroe, Lancs.

1904 Page, H. M., 14, Greville Place, S.W.

1901 Palmer, Frederick S., M.D., 7, Gordon Place, Gordon Square, W.C.

1906 Parker, Arthur Percy, B.Ch., Surgeon Radcliffe Infirmary; 2, Holywell Street, Oxford.

O.M. Parkinson, J. Porter, M.D., Physician at The London Temperance Hospital and Physician at The North-Eastern Hospital for Children; 57, Wimpole Street, W. (C. 1903–4, 1904–5, 1905–6.) (S.)

1902 Parkinson, Thomas W., M.D., 77, Sloane Street, S.W.

1903 Parry, Robert H., Surgeon at The Victoria Infirmary and at The Royal Hospital for Sick Children, Glasgow; 3, Royal Crescent, Queen's Park, Glasgow. (C.) (1903–4.)

1901 Paterson, Alexander Gordon, M.D., South Lodge, Ascot, Berks.

1903 Pearson, S. Vere, M.B., Physician at Mundesley Sanatorium, Norfolk.

1906 Peatling, Albert Victor, M.B., Park View, High Street, Carshalton, Surrey.

1901 Pedley, R. Denison, Dental-Surgeon at The Evelina Hospital for Sick Children; 17, Railway Approach, London Bridge, S.E.

1901 Pendlebury, Herbert S., B.C., Surgeon at The Royal Hospital for Women and Children, Assistant-Surgeon at St. George's Hospital; 44, Brook Street, W.

O.M. *Penrose, Frank, M.D., late Physician at St. George's Hospital and at The Hospital for Children, Great Ormond Street; Penmener, Parkstone, Dorset.

1901 Pernet, George, Assistant at the Skin Department, University College Hospital; 94, Harley Street, W. (C. 1903–4, 1904–5, 1905–6.)

Elected

1901 Perry, Herbert, M.D., Assistant-Physician at The General Hospital, Birmingham, Physician to Outpatients at The Children's Hospital, Birmingham; 54, Newhall Street, Birmingham. (C. 1905–6.) (C.)

O.M. Peskett, A. W. Chalmers, M.B., 4, Clermont Road, Preston Park, Brighton.

1902 Phillips, Hugh, M.B., 28, Palace Court, W.

1905 Pike, Douglas R., M.B., 3, Cromwell Crescent, Kensington, S.W.

1904 Piper, Francis Perris, M.B., King's Leigh, Tankerton, Whitstable.

1901 Poland, John, Senior Surgeon at The Miller Hospital, Greenwich; Surgeon at The City Orthopædic Hospital; 2, Mansfield Street, W. (C.)

1902 Potter, Bernard Elwell, M.B., Resident Medical Officer at St. George's Dispensary, Hanover Square; 57, Park Street, W.

1901 Potts, William A., M.D., 160, Hagley Road, Birmingham.

1904 Powell, Hugh Falkenberg, 1, Seagrove Place, Pittville, Cheltenham.

1904 Poynton, Frederick J., M.D., Assistant-Physician at University College Hospital, and at The Hospital for Sick Children, Great Ormond Street; 1, Harley Place, Harley Street, W. (C.)

O.M. *Prangley, Henry J., Tudor House, Anerley, S.E.

1901 Pratt, Eldon, M.D., Henfield, Sussex.

1901 Pritchard, George Eric Campbell, M.D., 14, Cromwell Place, South Kensington, S.W.

1901 Quennell, Arthur, Brentwood, Essex.

1905 Randall, Martin, M.D., The Lodge, Leopold Road, Wimbledon, S.W.

1901 Raw, Nathan, M.D., Mill Road, Liverpool.

1901 Ray, John Howson, Ch.M., Assistant-Surgeon at The Salford Royal Hospital; Surgeon at The Children's Hospital, Pendlebury; 11, St. John Street, Manchester.

1901 Rendel, Arthur Bowen, M.B., 43, Albion Street, Hyde Park, W.

1901 Rhodes, John Milson, M.D., Didsbury.

Elected

1905 Richardson, William G., B.S., Assistant-Surgeon at Newcastle-on-Tyne Royal Infirmary; 19, Saville Row, Newcastle-on-Tyne.

1903 Ridley, J. Brooke, M.D., 32, Sydenham Road, Croydon.

1906 Roberts, Charles, M.B., Surgeon at The Manchester Children's Hospital; 15, St. John Street, Manchester.

O.M. Robinson, Henry Betham, M.S., Assistant-Surgeon at St. Thomas's Hospital and Surgeon at The East-London Hospital for Children; 1, Upper Wimpole Street, W. (C. 1901–2, 1902–3, 1903–4.)

O.M. Rogers, Bertram, M.H., M.D., Physician at The Hospital for Sick Children and Women, Bristol; 1, Victoria Square, Clifton, Bristol.

1901 Rose, Samuel Frank, Assistant-Surgeon at The National Dental Hospital; Dental-Surgeon at The North-Eastern Hospital for Children; 59, Queen Anne Street, W.

O.M. Roth, Bernard, Orthopaedic-Surgeon at The Royal Alexandra Hospital for Children, Brighton; 38, Harley Street, W.

1902 *Roxburgh, Alex. Bruce, B.Ch., Surgeon at The Poplar Hospital, and Assistant-Surgeon at The London Hospital; 7, Henrietta Street, Cavendish Square, W.

1901 Rumboll, Sydney, Cavendish House, Leeds.

1904 Sale-Barker, Horace John Maurice Drummond, Medical Officer in Charge X-ray and Electrical Departments at Westminster Hospital; 45, Hill Street, Knightsbridge.

O.M. *Sansom, A. Ernest, M.D., Consulting-Physician at The London Hospital, and Consulting-Physician at The North-Eastern Hospital for Children; "Beversbrook," Finchley Road, Hampstead, N.W. (C.C. 1900–1, 1901–2.) (C. 1902–3.)

1904 Sawyer, James E., M.D., Casualty Assistant Physician at The General Hospital, Birmingham; Physician to Out-patients at The Children's Hospital, Birmingham; 93, Cornwall Street, Birmingham.

1904 Scofield, H. C. Lingham, M.B., "Northwood," Dulwich Grove, North Dulwich, S.E.

1904 Scott, W. J., M.B., Clifton House, Sheet Street, Windsor.

Elected

O.M. Sequeira, James H., M.D., Physician in Charge of the Skin Department at The London Hospital; 63, Harley Street, W.

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1901 Simson, Henry J. Forbes, 36, Grosvenor Street, W.

1901 Skelding, Henry, B.C., Surgeon at The Bedford County Hospital; St. Loyes, Bedford. (C. 1904–5, 1905-6.) (P.S.)

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1901 *Smith, Travers, R.M., M.B., 13, Kilburn Park Road, N.W.

1904 Smith, William R., M.D., Professor of Forensic Medicine, King's College, London; 74, Great Russell Street, Bloomsbury Square, W.C.

1901 Smyth, Sidney B., M.B., Physician at The Hospital for Sick Children, Belfast; Ulsterville House, Lisburn Road, Belfast. (C. 1903–4.)

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1903 Spriggs, Edmund Ivens, M.D., Assistant-Physician and Lecturer on Pathological Chemistry at St. George's Hospital, Assistant Physician to The Victoria Hospital for Children and at The City of London Chest Hospital; 48, Bryanston Street, Portman Square, W.

1904 Spurway, John, M.B., 79, Brondesbury Road, Kilburn, N.W.

1901 Staveley, William H. C., 24, Sloane Gardens, S.W.

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O.M. Sunderland, Septimus P., M.D., Physician at The Royal Hospital for Children and Women; Obstetric-Physician at the French Hospital; 11, Cavendish Place, W.

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1904 Swainson, James Midleton Graham, Assistant-Surgeon North-Eastern Hospital for Children.

1901 Swan, Russell H. J., M.B., 8, St. Thomas's Street, London Bridge, S.E.

1901 *Targett, James H., M.S., Assistant-Obstetric-Surgeon at Guy's Hospital; 19, Upper Wimpole Street, W.

1901 *Taylor, Frederick, M.D., Senior Physician at Guy's Hospital; Consulting-Physician at The Evelina Hospital for Sick Children; 20, Wimpole Street, W. (C. 1901–2, 1903–4.) (W.)

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1906 Telford, E. D., Surgeon, Children's Hospital, Manchester; 8, St. John Street, Manchester.

1905 Telling, W. H. Maxwell, M.D., 32, Park Square, Leeds.

1902 Thorne, Atwood, M.B., Assistant-Surgeon at The London Throat Hospital; 148, Harley Street, W.

1901 Thorne, W. Bezly, M.D., 2, Harley Street, W.

1902 Tod, Hunter, B.C., 111, Harley Street, W., Aural-Surgeon at The London Hospital.

1901 Trout, J. H. Ensor, 412, Monument Road, Birmingham.

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O.M. Tubby, Alfred H., M.S., Surgeon at Westminster Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; Surgeon at The National Orthopædic Hospital; 25, Weymouth Street, W. (C. 1900–1, 1901–2.) (C.C. 1902–3.) (C. 1904–5, 1905–6.) (C.)

1903 Tullis, George Augustus, M.D., Holmbush, Grove Road, Southsea.

1901 Turnbull, George Lindsay, M.D., 76, Ladbroke Square, W. (C.)

1901 Turner, H. Stanley, "Balgownie," 211, Bridge Road, Battersea, S.W.

1904 Turner, Philip, M.S., Assistant-Surgeon at The Belgrave Hospital for Children; 8, St. Thomas's Street, S.W.

1905 Tyson, W. J., M.D., Physician at The Folkestone Hospital; 10, Langborne Gardens, Folkestone.

O.M. Vacher, Francis, Consulting-Surgeon at The Wirrell Hospital for Children; "Glenmore," Shrewsbury Road, Birkenhead. (C.)

O.M. Vickery, William H., Surgeon at The Children's Hospital, Newcastle-on-Tyne; 52, Elswick Road, Newcastle-on-Tyne. (C. 1900–1.)

1902 Walker, J. W. Thomson, C.M., Assistant-Surgeon at The North-West London Hospital, Assistant-Surgeon at St. Peter's Hospital for Stone; 30, Queen Anne Street, W. (C. 1903–4, 1904–5, 1905–6.)

1901 Wallace, Joseph B., M.B., 39, Battersea Rise, S.W.

1905 Wareham, Sidney James, Surgeon, St. Mary's Children's Hospital, Plaistow; Raymond House, Plasket Road, Upton Park, E.

1901 Waring, John Alfred, M.B.

1905 Webb, F. C. Apthorpe, Grafton House, Cambridge.

1901 *Weber, F. Parkes, M.D., Physician at The German Hospital, Dalston; Assistant-Physician at The North-London Hospital for Consumption; 19, Harley Street, W. (C. 1905–6.)

1902 Westmacott, Frederick H., Aural-Surgeon at the Manchester Children's Hospital, Pendlebury; 8, St. John Street, Manchester.

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1901 **Whipham, Thomas Rowland C.**, M.D., Physician to Out-patients at the Evelina Hospital for Children: 37, Park Street, W.

1906 **White, Sinclair**, M.D., Ranmore, Sheffield.

1901 **Whitelocke, Richard H. A.**, C.M., Surgeon at The Radcliffe Infirmary, Oxford; 6, Banbury Road, Oxford. (C. 1901–2, 1902–3, 1903–4.) (P.S. 1904–5, 1905–6.) (C.)

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O.M. **Williams, Edward Cecil**, M.B., Physician to Out-patients at The Bristol Royal Hospital for Sick Children; 9, Whatly Road, Clifton, Bristol.

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1904 **Wilson, Geoffrey R.**, Ongar, Essex.

O.M. **Wilson, Kemlo R. M.**, M.D., Hanwell, W.

1905 **Wilson, R. Mervyn**, M.D., Surgeon, Derbyshire Hospital for Sick Children; Full Street, Derby.

1902 **Wood, Edward**, Stamford Lodge, Waverley Road, Enfield, Middlesex.

1901 **Woods, John Francis**, M.D., Medical-Superintendent at The Hoxton House Asylum, N.; 29, Queen Anne Street, W.

1901 **Wright, Douglas**, M.D., 229, Camden Road, N.

1902 **Wright, George A.**, M.B., Surgeon at The Manchester Royal Infirmary; Surgeon at The Children's Hospital, Pendlebury, Manchester; 16, St. John Street, Manchester.

1904 **Wylie, Andrew**, M.D., 7, Harley Street, W.

⁂ *It is requested that any inaccuracy in the foregoing list may be notified to the Honorary Editor of the Reports and also to the Senior London Honorary Secretary.*

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REPORTS

OF

THE SOCIETY

FOR THE

STUDY OF DISEASE IN CHILDREN.

SESSION of 1905-6.

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1. UNILATERAL AND BILATERAL PERSISTENCE OF THE FOURTH BRANCHIAL CLEFT.

By P. LOCKHART MUMMERY, B.C.

Case 1.—The patient is a girl, aged 8 years. There is a minute opening on both sides of the neck just at the anterior border of the sterno-mastoid muscles and about three quarters of an inch above the sterno-clavicular joints. From the opening on the right side a sinus extends upwards to a point behind the tonsil. It is not possible to make certain whether it communicates with the pharynx or not. Closely associated with the sinus in its whole length, there is a hard bar of cartilage which can be distinctly felt under the skin.

On the left side the sinus can only be traced for about two inches, and just behind the external opening there is a cyst about the size of a hazel nut.

From the opening on the right side there is a discharge of clear mucus, while from the left opening there is a milky discharge. When the child eats or cries there is a considerable increase in the amount of discharge from the openings. The openings in the neck were first noticed when the child was ten months old. In this case an hereditary factor can be traced through two generations.

Case 2.—The patient is a boy, aged 9 months. There is a small opening in the skin on the right side of the neck at the anterior border of the right sterno-mastoid muscle, and about half an inch above the right sterno-clavicular point. From this opening a thick cord can be traced upwards beneath the skin for about three quarters of an inch, and ending above in a cyst which is situated beneath the sterno-mastoid muscle. Pressure on this cyst causes a yellowish discharge to escape from the opening below.

The condition was first noticed when the child was four months old.

(*Exhibited October the 20th*, 1905.)

Discussion.

The Chairman (Dr. Percy Lewis) (Folkestone) asked whether the discharges had been examined microscopically, and if so were they salivary or mucous? He would also like to hear how he accounted for one being clear and the other thick. With regard to the structure which was thought to be cartilage, had Mr. Mummery considered the possibility of it being only a lymphatic gland? On a cursory examination it did not seem to him to be a cartilage.

Mr. Lockhart Mummery, in reply, said he thought the explanation was that when there was a complete fistula the inner part was lined with mucous epithelium, similar to that of the primitive pharynx, while the external portion was lined with epidermal tissue. Therefore if the sinus were nearly persistent throughout, the discharge was mainly mucus from the back part, whereas if the sinus were a short one the discharge was mainly epidermal. On the left side there was a cyst in connection with the sinus, which he thought was secondary, and in which there was probably secondary infection, because pus came from the left side. After careful examination he concluded that the lumps were cartilage owing to their situation, and not glands. They seemed to be intimately attached to the sinus and to lie superficial to it. The treatment he proposed to carry out was to dissect out the sinuses. He had done that in a case he previously had and the sinus came away easily, with the exception of the extreme inner end, which was very difficult to remove. The chief reason for removal was that very much later in life a certain number of those abnormalities became sarcomatous or epitheliomatous.

2. A CASE OF LYMPHADENITIS WITH ENLARGEMENT OF THE SPLEEN AND PECULIAR BLOOD-CHANGES.

By T. R. WHIPHAM, M.D.

The patient is a girl, aged 2 years and 2 months. The family history is good, except that the mother is delicate. The patient has three healthy sisters, but one other sister and two brothers have died from bronchitis.

The patient herself had bronchitis as a baby and measles one year ago. She was apparently quite well till August the 21st, when enlargement of the glands was first noticed, fretfulness and anorexia being the only other symptoms. A week later she was brought to my out-patient department at the Evelina Hospital. She looked ill and the temperature was 100·5° F. Enlarged glands were felt in the cervical, axillary, and inguinal regions. The liver did not appear to be unduly large and the spleen could not be felt with certainty. No enlargement of the thymus was made out. The following week the child was in much the same condition, but the temperature was 101·8° F. At the third week the glands in the groins were found to be larger, and fresh enlargements could be felt in both femoral and in the left axillary regions. The liver was moderately enlarged and the spleen could be definitely felt below the ribs. The temperature was not so high as before, but the child was evidently worse, and could scarcely walk, so I admitted her into the wards. In the course of the next few days the glands still further increased in size. The first examination of the blood was made a fortnight after the onset of the symptoms and showed—red cells, 4,736,000 per c.mm.; white cells, 25,000; hæmoglobin 84 per cent. The differential count, according to my own observation, was—polymorphonuclears, 2·4 per cent.; large lymphocytes, 65·9 per cent., small, 30·5 per cent.; transitionals, 1·2 per cent. Three nucleated red cells (normoblasts) were found to 500 white (0·6 per cent. of white cells). The house-physician (Dr. Gompertz) made a separate count from another film and found an almost identical number of mononuclear cells, though there was some discrepancy between the two counts as to the numbers of large and small lymphocytes. Dr. Gompertz also found 0·4 per cent. of eosinophiles. These observations were subsequently verified by Mr. Leathem, the hospital pathologist, and on September the 25th he reported—leucocytes, 17,400 per c.mm.; polymorphonuclears, 2 per cent.; large lymphocytes, 80 per cent., small, 14 per cent.; large mononulears, 3·2 per cent.; transitional cells, 0·4 per cent.; myelocytes, 0·4 per cent. Normoblasts, 0·8 per cent. of white cells. The temperature, which had never been steady, about this time became very irregular, reaching 101° F. and over 102° F. On September the 26th it shot up to nearly 105° F. coincident with the eruption of a severe attack of herpes zoster on the left side of the chest. It soon fell, however, and has subsequently remained normal, the glands at the same time showing a tendency to decrease in size. The proportion of polynuclear cells in the blood has also slightly increased, there being 10·63 per cent. on October the 5th and 15 per cent. on the 13th.

(*Exhibited October the* 20*th*, 1905.)

Postscript.—Death occurred on November the 12th.

3. A CASE OF SPLENIC ANÆMIA?

By T. R. WHIPHAM, M.D.

The patient is a girl, aged 8 years, who is very small for her age and presents a marked grade of anæmia.

Her mother is anæmic and one of three younger children has a chronic cough; otherwise there is nothing in the family history.

The child herself has always been delicate and attended the Evelina Hospital for wasting and rickets during the first eighteen months of life. When three years old she had an attack of pneumonia and shortly afterwards measles, which was followed by severe diarrhœa. She continued in fair health, though always weak and delicate, till about six years of age, when her mother noticed that she began to get gradually paler.

She was first admitted into the Evelina Hospital, under the care of Dr. Willcocks, in October, 1904, with a temperature of over 103° F. and marked anæmia, the red cells numbering only 816,000 and the white 3400 per c.mm. The hæmoglobin was reduced to 20 per cent. The polymorphonuclear cells were 55 per cent. and the lymphocytes (large and small together) 37 per cent.; 4·5 per cent. of basophiles were also found. The abdomen was very distended and the spleen could just be felt. She rapidly improved under arsenic and iron, all the elements of the blood being doubled after a fortnight, but she was troubled, and has been so all through her illness, with frequent vomiting, which has been proved unconnected with the administration of arsenic. She was sent to Brighton for a month, but was readmitted into hospital at the end of that time with severe sickness and a slight deterioration in the quality of the blood. The temperature was irregular, rising at times to 103° F., but the rises were unaccompanied by any disturbance. No enlargement of the spleen at this time could be made out. She again improved somewhat, and on being discharged I kept her under observation as an out-patient. In February of this year she had 3,760,000 red cells, hæmoglobin 72 per cent., and 8600 white cells per c.mm., the various kinds of the latter being in practically the same proportions as when she was first seen. In April last she was again admitted into hospital with more sickness and the same irregular temperature. The red cells had fallen to just over 2,000,000 and the hæmoglobin to 32 per cent. The spleen could then be felt two inches below the ribs. After two months in the hospital she was again discharged, having shown no improvement in her blood, and was subsequently sent again to the seaside. In

September her mother was summoned to the home to fetch her away as she was much worse, and brought her straight up to the hospital, where I happened to be at the time. The child looked almost bloodless and was much collapsed after the journey. She was taken in under my care, and a blood-count showed only 827,500 erythrocytes and hæmoglobin 15 per cent. The leucocytes were 9200 in number, 61 per cent. being polymorphonuclears, 21·6 per cent. small lymphocytes, and 7 per cent. large. The red cells showed great variation in size, poikilocytosis and polychromasia and two normoblasts per 100 white were found; the spleen could just be felt. Under a further course of arsenic and iron the child has again improved, the red cells and hæmoglobin being nearly doubled after three and a half weeks. The last count, made two days ago, shows 1,480,000 red cells, hæmoglobin 28 per cent., and 4000 leucocytes.

The spleen at the present time cannot be definitely made out; there is no glandular enlargement and never has been. The child is thin and pale, but not so anæmic as she was a month ago. Her weight is 2 st. 1½ lbs., and has increased only by 2 lbs. in the last eleven months.

She is bright and cheerful, but is frequently very sick without any accompanying nausea. The abdomen is large and soft and the liver is just palpable. In the chest and neck are well-marked hæmic bruits.

(*Exhibited October the 20th*, 1905.)

Postscript.—The patient died on December the 5th. Gastro-enteritis and catarrhal appendicitis were found post-mortem, but no sign of tubercle was present.

4. SPLENOMEGALY IN A BROTHER AND SISTER.

By J. PORTER PARKINSON, M.D.

Ernest N—, now aged 11 years, was admitted first into the North-Eastern Hospital for Children under my late colleague Dr. Sequeira in September, 1899. His history was that he had always been pale; in his mother's expressive language, "he never had a drop of blood in his body," his abdomen had been large since he was a year old, and at that time a doctor had told her his spleen was enlarged. He had occasional swelling of the feet and legs, and shortness of breath. There was no history suggesting congenital syphilis. At this time he weighed $38\frac{1}{2}$ lbs., he was anæmic, and there were purpuric spots on the neck, chest, and legs. The temperature was normal. The heart and lungs were normal. The abdomen was enlarged and bulging on the left side owing to the splenic enlargement, which extended from the seventh rib in the mid-axilla to half an inch below the anterior superior iliac spine. The liver extended two fingers' breadth below the costal margin in the right mammary line. There was no ascites. The urine was normal. Examination of the blood showed—hæmoglobin 25 per cent, red corpuscles 2,960,000 per c.mm.; there were many microblasts, no megaloblasts, and there was much variation in size of these cells; white corpuscles 25,000, polymorphonuclears 75 per cent., small lymphocytes 17·5 per cent., large lymphocytes 7·5 per cent., eosinophiles 2·5 per cent., myelocytes 0·5 per cent.

Under treatment with iron, etc., the hæmoglobin and red corpuscles increased markedly, and the child improved in every respect, except that the spleen was not reduced in size.

The child was readmitted under my care October the 2nd, 1905, with the history of having been in good health since discharged from the hospital, but for the past month there had been nocturnal enuresis, which stained the bedclothes more than usual. The boy was pale, and there was a slight tinge of jaundice in the skin and conjunctiva.

The lungs were normal and the heart not enlarged, but there was a systolic murmur, heard best at the pulmonary cartilage. The abdomen was full and the spleen was much enlarged, the dulness above extending to the sixth rib in the mid-axillary line and below it could be felt reaching below the anterior superior iliac spine; it was smooth and hard, with two well-marked notches in its anterior edge.

The dulness of the liver extended upwards to the fifth rib in the mammary line, and its lower edge was felt two fingers' breadth below the costal margin. There was no evidence of ascites. There was no enlargement of the external lymphatic glands, and the tonsils were healthy.

The examination of the blood showed—60 per cent. hæmoglobin, red corpuscles 2,966,000 per c.mm., white corpuscles 15,000, polynuclears 76 per cent., small mononuclears 20 per cent., large mononuclears 2 per cent., polynuclear eosinophiles 2 per cent. There was no definite abnormality of the red cells; a few were slightly smaller than others, and in very rare instances a pear-shaped corpuscle, but nothing beyond what might be seen in the blood of any patient.

The younger child, aged 1 year, has a lemon-yellow skin, but no jaundice; it showed marked signs of rickets, the fontanelle being very large, and the general shape of the head is that of rickets; the ribs are beaded, and the thorax shows well-marked rickety deformity; five teeth are present. The spleen is much enlarged, extending below to the anterior superior spine of the ilium, and its upward dulness reaches to the seventh rib. The liver can be felt down to one finger's breadth below the costal margin.

The blood examination showed—erythrocytes 3,393,000 per c.mm., leucocytes 52,570, polynuclears 42 per cent., eosinophiles 10 per cent., small mononuclears 36 per cent., large mononuclears 9 per cent., myelocytes 3 per cent. (Ehrlich's triacid stain). There is some variety in size of the red corpuscles, but none are very large, and there is very little poikilocytosis, not more than usual in infants under the age of two years. The mother has had thirteen children, seven of whom are living. She has had no miscarriages. The causes of death of the others were as follows: two years, "tumour of bowels"; twelve months, "consumptive bowels"; eighteen months, "inflammation of bowels"; two months, wasting; three months, fits; three months, wasting. The other children are alive and stated to be healthy.

It is difficult to say whether these two cases belong to the group known as "splenic anæmia" or those called by the cumbrous name "metaspleno-megalic hypertrophic biliary cirrhosis," in which enlargement of the spleen is the earliest sign and of which Gilbert and Fournier described several cases in young children.

The blood-count is not characteristic of the former, for at the present time the colour index is normal and the leucocytes appear to have been constantly increased in number, whereas leucopenia is the rule in splenic anæmia. Moreover jaundice does not occur in splenic anæmia, while slight jaundice has lasted for some weeks in this boy and therefore appears not to be an accidental complication. This jaundice has not been accompanied by pain, which was a marked feature in two cases shown by Sir T. Barlow and Dr. Batty Shaw at the Clinical Society in 1902.

The only form of hæmorrhage which the boy has had is the slight purpura noted when in the hospital six years ago.

(*Exhibited October the 20th*, 1905.)

5. A CASE OF SPLENOMEGALY.

By EDMUND CAUTLEY, M.D.

Alice T—, aged 8 years, was admitted into the Belgrave Hospital for Children on September the 5th, 1905, for occasional epigastric pain, during the previous two months, and enlargement of the abdomen. Family history: Fourth of six children. One died of croup and the others are healthy. The parents are healthy. The mother has had three miscarriages. Past history: She was nursed for six months; had whooping-cough, chickenpox, and measles during the first five years of life; had never been abroad. On admission she was well nourished, with a certain degree of yellowish pallor and general anæmia. The tongue was furred and the teeth very carious. No enlarged glands were found. A systolic hæmic murmur was present over the cardiac area.

The abdomen was large and prominent. The spleen extended to the middle line and as low as the level of the umbilicus. It felt very hard and smooth.

The liver extended an inch and a half below the costal margin.

A differential leucocyte count on September the 9th showed—lymphocyctes 68·0, polymorphonuclear cells 29·2, eosinophiles 2·0, and basophiles 0·8 per cent. The total white cells on September the 11th amounted to 2600 and on the 17th to 4000 per c.mm. On the latter day a few myelocytes were found.

On October the 16th the number of red cells was 4,050,000 per c.mm., white cells 5450, and hæmoglobin 70 per cent. Differential count: small lymphocytes 50·0, large lymphocytes 11·3, polymorphonuclears 34·0, and eosinophiles 2·0 per cent.

The child's general condition has improved, the anæmia is less, and the spleen is distinctly smaller. The abdomen is still very large and the liver is unaltered. She has gained weight, increasing from 57 lbs. 6 oz. on September the 6th to 58 lbs. 12 oz. on September the 23rd, and 61 lbs. on October the 17th, a gain of 3½ lbs. in less than seven weeks. There is still a hæmic cardiac murmur. She has been fed on a liberal diet and given dialysed iron.

This case appears to belong to the class of splenic hyperplasias sometimes called von Jaksch's disease, or anæmia splenica infantum. It is, however, impossible to be certain of its nature; a maternal history of three miscarriages is suspicious.

The difficulty of accurate diagnosis in cases of splenic enlargement in children was well illustrated by another case under observation at the same time. The child, a girl aged 17 months, was very thin, weighing only 11 lbs., anæmic, very feeble, and had only cut six teeth, which were already carious. Râles were heard in the chest. The spleen extended below the level of the umbilicus and the liver almost as low. The eyelids and feet were œdematous. Death resulted from asthenia five days after admission into hospital. A family history of phthisis and meningitis was obtained.

At the post-mortem examination the lungs were found riddled with tubercles. The bronchial and cervical glands were tuberculous. The liver weighed 12 oz. and was somewhat fatty. The spleen weighed 3½ oz. and was full of tubercles. Tubercles were found in the kidneys and liver. The mesenteric glands were enlarged and some of them tuberculous.

Thus, the cause of the splenic enlargement was tuberculosis, and death was due to general miliary tuberculosis. The condition was suspected on account of the family history, the wasting, the lung signs, and the œdema.

(*Exhibited October the 20th,* 1905.)

Discussion.

The Chairman (Dr. Percy Lewis) (Folkestone) desired to hear what was the prognosis in the cases mentioned, and what the readers of the papers thought about tubercle as a cause. Syphilis seemed to be ruled out of court in all the cases. Dr. Whipham's second case, which he (Dr. Percy Lewis) had examined carefully, looked very much like tubercle to him, and he would have been inclined to regard it as an instance of tabes mesenterica. The abdomen was distended, and the aspect gave one the idea of that disease; in addition the lumps usually associated with it were to be felt. With regard to treatment, Dr. Whipham mentioned arsenic and iron, but treatment had not been referred to in connection with the other cases.

Dr. Robert Hutchison said there could not be a better illustration than had been afforded at that meeting of the extraordinary difficulty and complexity of such cases. Anæmia in children was plain sailing enough until one got to the splenic group, and when one found splenic hyperplasia of any sort one became confused. He did not think much progress would be made in the diagnosis of such cases so long as it was ordained that they should be grouped entirely on clinical signs. What it was necessary to know was the etiology; a classification based upon causation was the only satisfactory one, because anæmia, due to totally different causes, seemed in children to be very apt to give rise to enlargement of the spleen. That enlargement occurred apparently as a purely secondary event. Really it should not be allowed that degree of diagnostic importance which was assigned to it. The two cases shown by Dr. Whipham were extremely interesting, especially the first one, and it was so because of the unusual character of the blood-count. The chief feature of it was that there was a very large percentage of mononuclear cells, the polynuclears remaining comparatively low in number. An additional difficulty was introduced into cases of that sort by the fact that it had recently been found that many of the so-called large mononuclear leucocytes which had been considered to belong to the lymphocyte group were non-granular myelocytes, and that they would eventually become such in full development. He would regard that case as one of the mixed-cell leukæmias, one which had been regarded as intermediate between the lymphatic group of leukæmias and the myelocytic group. Probably a large number of the mononuclear cells were non-granular myelocytes derived from bone-marrow. A considerable number of such cases had been described, and they appeared specially apt to occur in children in whom the blood-forming processes were not so stereotyped as in grown-up persons. With regard to Dr. Whipham's second case, he would regard that as severe secondary anæmia with a merely accidental enlargement of the spleen. He thought that must be classed as a secondary anæmia because of the way in which the red cells were specially hard hit, the polychromatophilia indicating a severe degree of destruction. That might be secondary to chronic gastro-intestinal disturbance, or it might be that the case was tubercular, with secondary enlargement of the spleen. The most interesting cases of the group were those of Dr. Porter Parkinson. There had been described a form of splenic enlargement along with liver enlargement, which was spoken of as the Grancher type of splenic enlargement. It was very rare, but it occurred in families. The pathological lesion was a definite one, namely an overgrowth of the endothelium of the spleen, so that the spleen was converted into a large endothelioma, and there was also a secondary endotheliomatous overgrowth in the liver. One would be tempted to refer the case of the boy to that group, and that might explain the jaundice. In the case of the younger child the condition seemed very suggestive of syphilis. True, there were no miscarriages, but the family history was bad in regard to early deaths; and if he had looked at the younger child alone he would have thought it was a case of splenic anæmia with a syphilitic taint. In Dr. Cautley's case he thought the characteristic feature was leucopenia, and that was the characteristic point in the splenic anæmia of the adult type. But, again, one was met with the difficulty that chronic splenic enlargement from any cause was apt to be associated with leucopenia. He did not know why that was, but if one had a case of undoubted syphilis, with notched incisors, and a definite syphilitic history and a chronically enlarged spleen without any obvious anæmia, one would almost certainly find in it a diminished number of white cells; and there was a curious association between enlargement of the spleen from any cause and diminution of the white cells. On the whole, he would refer that case more to the class of the adult type of splenic anæmia than to anything else, but one could not exclude the possibility of it being a syphilitic overgrowth, and that the leucopenia was the result of chronic enlargement of the spleen, which would be found to be a fibrous overgrowth. He thought the enlargement of the spleen had not to be taken into account at all in considering the prognosis. So far as he could discover, the prognosis depended much more on the heteromorphous white cells; the more one found an aberrant type of cell the worse the prognosis was. Many of the chronic cases persisted for years and were ultimately recognised as the so-called splenic anæmia of adults, or Banti's disease. One such case was reported by Dr. Rolleston which came under his observation at St. George's as a young man; but it was found that the child had been in Gt. Ormond Street Children's Hospital for enlargement of the spleen and anæmia, and no doubt the condition had persisted all those years. With regard to treatment, he did not think iron and arsenic had anything like the same specific influence in those cases of enlarged spleen as it had in ordinary secondary anæmia. These drugs could only stimulate the bone-marrow; and the reason they acted so much less effectively in children than in adults was, that nearly the whole bone-marrow in the child was of the red type, and it was using its bone-marrow to form blood up to the limit of its capacity, and it could not be stimulated to develop more; whereas in adults there could be some replacement of the yellow part of the marrow by red, and the secondary anæmia might be compensated in that way. He thought the only effective treatment was attention to the general nutrition, putting any disorder of stomach and bowels right, and carrying out ordinary hygienic treatment with fresh air and sunlight. He would like to see the X rays tried for pseudo-leukæmia. So far as he knew they had not been tried in such cases, and it would be interesting to see whether the chronic splenic enlargement could be influenced favourably by such treatment.

Mr. L. S. Dudgeon thought that the classification of splenic anæmias to be of any value must be on a pathological basis. Examination of the records of cases which had been published showed that the pathological side of the question had been very much wanting. Even many of the blood examinations had been insufficient. In the post-mortem room, in cases of splenic anæmia of children, one might meet with many different varieties. A very interesting class, and one to which few people referred, was the hæmolytic type. Two cases might appear to be identical, even in the post-mortem room, but examination of the liver and other viscera would show marked hæmolysis in one instance, whereas an exactly similar case seen during life would give no evidence of hæmolysis. Another point was the examination of the glands to which attention had lately been drawn, namely the hæmolymph glands. They were large succulent glands lying along the vertebral column and along the course of the aorta; they contained large blood-vessels, as the name implied. Nucleated red cells, various forms of myelocytes were found in these glands; in fact, their structure in some cases was similar to that of the bone-marrow. There was another type of splenic anæmia in which there was no evidence of these hæmolymph glands. Examination of the bone-marrow in children was not so important as in adults, because, as Dr. Hutchison had said, the bone-marrow is red in the shaft of the long bones in children and very little difference can be made out in many instances in the various anæmias of children. One speaker (Dr. Hutchison) said the cells were really "myelocytes" and not lymphocytes in the blood in some instances. That question had frequently cropped up at the Pathological Society of London, but there was no positive evidence in favour of this special type of cell being classed as myelocytes. If we could diagnose leukæmia by the presence of neutrophilic myelocytes alone, this question raised by Dr. Hutchison might have some support, but this is impossible. Enormous numbers of nucleated cells, such as were found in no other disease, mast-cells in large numbers, and various types of myelocytes are present in the blood in myelæmia, without exception; neutrophilic myelocytes are never found alone. It was stated when he (Mr. Dudgeon) read a paper on lymphocythæmia at the Pathological Society about six months ago, that the cells in the blood in his case were really neutrophilic myelocytes, and if Ehrlich's stain was used that fact would be proved. He had used Ehrlich's stain for a long time, and he tried it again on the blood-films in a case of lymphocythæmia and found they were mononucleated cells without evidence of granules, exactly similar results to those obtained with Leishman's stain. He did not think these non-granular cells should be regarded as myelocytes until actual proof was forthcoming.

Dr. Theodore Fisher said, in reference to the prognosis of such cases as those under discussion, that he had looked up cases of splenic anæmia of infancy which had been under his care, and they were all well at the age of two years. They were comparatively healthy children. Tuberculosis of the spleen occurring alone was well known to be an extremely rare condition. As in Dr. Cautley's case, very occasionally enlargement of the spleen due to tuberculosis might be an early feature of abdominal tuberculosis.

Dr. T. R. Whipham, in reply, said he was not sure of his own diagnosis in his second case, and he thought Dr. Hutchison's diagnosis of anæmia secondary to gastro-intestinal disturbance was the most likely alternative one. If it were a case of abdominal tuberculosis, as suggested by the Chairman, why should there be such marked anæmia? And in tuberculosis of the glands he believed there was generally a lymphocytosis. With regard to the other case, should it turn out eventually to be a case of myelogenous leukæmia, he trusted he might be allowed to report the fact to the Society.

Dr. Porter Parkinson, in reply to the suggestion that congenital syphilis was a factor in one or both of his cases, said the mother had thirteen children but no miscarriages whatever. A number of the children had died young, but that was not at all an unlikely occurrence in a family of that class where children came rapidly, quite apart from syphilis. Dr. Hutchison later on had stated that those cases of congenital syphilis with enlarged spleen usually had no leukæmia. The younger child had 52,000 leucocytes to the c.mm., and that was a distinct leucocytosis, quite four times the usual number for the age. It was generally considered that arsenic did not do much good in those cases, and some people thought that the arsenic might have produced the cirrhosis of the liver which was occasionally found in post-mortem examinations.

Dr. Edmund Cautley, in reply, pointed out that in his case the treatment consisted of liberal feeding and moderate doses of dialysed iron, to which he did not attach much importance; but he did attach importance to the removal of five carious teeth. He was strongly convinced that the chief cause of the anæmia, and probably of the splenic enlargement, was the toxæmia from decayed teeth. The child had gained $3\frac{1}{2}$ lbs. in less than seven weeks, therefore he did not consider there was any serious disease present, and the prognosis was excellent.

6. CONGENITAL TUMOUR OF THE ARM, PROBABLY LIPOMA.

By W. MILNER BURGESS.

The patient, a girl, aged $3\frac{2}{12}$ths years (the second child), was born on August the 6th, 1902, the labour being normal. The mother was anæmic and eczematous.

The tumour was noticed three weeks after birth on the middle third of the right arm, on its anterior surface, and consisted of three small lobes the size of peas. It was movable and painless, and was placed between the coraco-brachialis and biceps muscles.

On November the 7th, 1902—that is, when three months old—the tumour was the size of three horse-beans, at which time the infant was suffering from whooping-cough.

On September the 30th, 1905—that is, at $3\frac{1}{12}$th years old—the tumour was lobulated and the size of three Spanish nuts and said to be growing. The skin over it had a yellow, bruised appearance, as if the tumour had been squeezed by accident.

There was no history of any such tumour in her family.

On October the 14th, 1905, all the bruising had gone. The tumour was probably a lipoma.

(*Exhibited October the 20th*, 1905.)

7. MONGOLIAN TYPES—EAST AND WEST.

By G. A. SUTHERLAND, M.D.

Dr. G. A. Sutherland showed two Mongolian types—(1) a pure Japanese female infant, aged 10 months; and (2) a Mongolian imbecile, aged $2\frac{1}{2}$ years.

He referred to the name "Mongolian imbecile," which was used because of certain facial appearances in this form of imbecility which were suggestive of a Mongolian origin. The small and sloping palpebral fissure, the epicanthus, and the depressed bridge of the nose were the special facial similarities which had suggested the name. Imperfect development of the bones at the base of the skull was probably responsible for these appearances. Other defects in development were frequently found in other parts of the body. The mental defect was apparent from a very early age.

While the Mongolian imbecile of English extraction was to be regarded as a pathological product, the Eastern Mongolian was the physiological product of a pure-bred race. The Japanese baby, however, possessed certain features of anthropological interest, in the shape of blue pigment spots scattered over the back, and specially well marked in the sacro-gluteal region. These pigment spots were well seen in the baby shown, there being a large blue patch over the buttocks, and some smaller spots over the back and shoulders, and one on the dorsum of the left hand. Regarding this pigmentation, it was stated that it might increase in extent for some months after birth, but usually disappeared entirely during the first few years of life.

(*Exhibited October the 20th,* 1905.)

Discussion.

Dr. George E. Shuttleworth (Richmond) said it was extremely interesting to him to see a real Mongol beside the Mongolian imbecile exhibited that evening. The pigmentation to which Dr. Sutherland had alluded was an entirely new feature to him. He could not give any information as to books in which reference was made to the subject, but with regard to the Western form of Mongol imbecility, he had observed nothing approaching to the pigmentation; still, it was a point which in future one would take care to investigate, and perhaps look through past cases in which one had not taken the precaution to examine carefully into anything of the kind. A passing thought was that there might be some negro blood, some atavism, showing itself in this pigmentation in the Japanese. With regard to the downy growth on the forehead to which Dr. Sutherland had alluded as existing with the normal Japanese child, he could not help thinking that it did exist, apart from any syphilitic origin, in a considerable number of specimens of Mongolian imbeciles. He could show Dr. Sutherland three or four of such cases, and this downiness existed in several cases of whom Dr. Cockburn would show portraits. In those cases he believed there was no suspicion of syphilitic history. He had previously compared notes with Dr. Sutherland with regard to the influence of syphilis in the etiology of Mongol imbecility, and it had been his own experience not to have ascertained the existence of inherited syphilis in any large number of Mongols. In fact, the only instance he was perfectly certain of happened to have been in a former patient of Dr. Sutherland's, who had come into his hands.

Of course, syphilis might be a cause No doubt any debilitating condition might be a potent factor in the production of that particular form of degeneration or retrogression, harking back from Caucasian characters to a more unfinished type. But there was one case that recently came under his notice which strongly impressed him with regard to the etiological action of maternal debility. The father was a vigorous Dutch gentleman, a University graduate, and the mother a woman of obviously good intelligence and health, and she brought her child to him when it was 5½ years of age. Her account was that when she became pregnant for the first time it was ascertained that she had a small pelvis, and she was recommended to place herself in the hands of a lady doctor in an Amsterdam hospital previous to the birth of the child. The treatment (extending over four or five months) consisted of practically starvation diet, the view presumably being that if the fœtus were underfed it would be a small one and consequently more viable. She went on to the full period of gestation, the labour was difficult, and instruments were used, but he did not think the instruments had anything to do with the condition. The child came into the world a characteristic "Mongol." There was no tubercular or syphilitic history in the family, and he could not help thinking that the debilitating course of treatment to which the mother was subjected had an influence in determining the type. Still, it was curious that it should be that particular form of imbecility. He had looked to see if in the Japanese baby shown there were any particular form of the hand, such as is characteristic of the Mongol imbecile, but could not find any. There was no incurvation of the little finger, and the fingers were not stumpy. And in Mongol imbeciles one often found between the great toe and the next toe a cleft, and the toes very much of the same length. But the Japanese child was quite normal in these respects. Of course, the peculiar mental condition depended essentially on the brain-formation, and it had been observed by Dr. Wilmarth and others that in the imbecile "Mongol" brain the medulla and the pons, and sometimes also the cerebellum, were disproportionately small as compared with the cerebrum, and the contracted base of skull might be the reason of the obliquity of the orbital plates and palpebral fissures. He would be glad to hear where he could find a good anatomical description of a normally-developed brain of a Japanese or Chinese. Dr. Mott had shown a photograph of the brain of a Chinese coolie who had fallen a victim to beri-beri, and the convolutions in that seemed very much like those of a "Mongol" brain; they were comparatively coarse and simple. But Dr. Mott told him he had now several Chinese brains for examination, and probably more would be heard on that subject shortly. Dr. Mott wrote in regard to them—"I have been struck by the complex convolutional pattern of the six brains I have received and their good size." Therefore it would seem that the brain of the Chinaman or Japanese was not really comparable as regards simplicity with that of the Mongol imbecile; it might be that in the true Mongolian the convolutions were somewhat differently packed in European and in Mongolian skulls.

Dr. R. P. Cockburn said that was his first acquaintance with a Japanese baby, and it was interesting to compare in the two children the flexibility of the limbs and softness of the tissues of the Mongolian imbecile as contrasted with the normal firmness of the Japanese child. He had photographs of a (Mongolian) mentally deficient child, showing a sulcus, which occurs between the great toe and the other toes in many of these children, but to which little attention has been directed. The sulcus is absent in the child shown this evening, and it perhaps only appears when the children begin to walk. It was also interesting to notice the different expression of the emotions in the two children. In the Japanese there was an average amount of control over the expression of the emotions, whereas the deficient child showed the characteristically exaggerated expression of the emotions very distinctly. The tongue only became fissured at about two years of age, and it was interesting to see the fissures beginning to appear in the imbecile child.

8. A CASE OF DIFFUSE ANGIOMATOUS CONDITION OF THE LIVER.

By JAMES E. H. SAWYER, M.D.

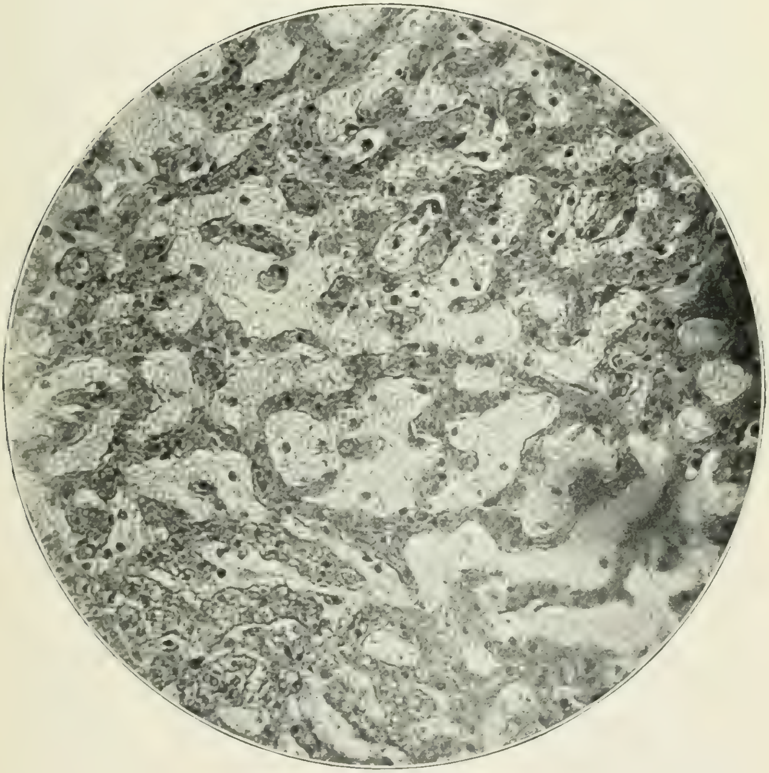
By the kind permission of Dr. Simon I am enabled to publish the following case.

Harold S—, aged 7 years, was admitted into the General Hospital, Birmingham, complaining that the abdomen had gradually been increasing in size for fifteen months.

Family history.—The father is alive, but is described as being very delicate, and from the history that the mother gives there seems very little doubt but that he is suffering from phthisis. The mother is in good health and has had no serious illness. There have been eleven children, six of whom are alive and quite healthy. Four children died in infancy from "convulsions," and all of these were under one year of age. There was one miscarriage. No history of syphilis can be obtained.

The child was never quite as healthy as the other children, and always seemed feeble. He was fed at the breast until twelve months old. There was never any sign of rickets, and he was not backward in any way, but he often complained of feeling tired and did not play about as other children of the same age. He had none of the specific fevers. The boy said that his mother gave him a "drop of beer," but this was on one day only. About three years ago he was treated at the hospital for ulceration of the mouth. About fifteen months ago his mother first noticed that his abdomen was becoming enlarged, and it has gone on increasing in size ever since, but much more rapidly during the last six months. At times he suffered from a slight degree of jaundice, and occasionally he had epistaxis. There was never any vomiting.

On a previous occasion he was admitted into the hospital under Dr. Simon, in August, 1904, and at that time the swelling of the abdomen had been noticed for about three months. The chief symptoms that were present then were pains in the upper part and enlargement of the abdomen, and a constant feeling of hunger. The bowels were opened about four times a day. On examination on his first admission into the hospital, the liver was found to be considerably enlarged, extending about three inches below the costal margin in the right nipple line. The liver seemed to be quite smooth, its lower edge could easily be felt, and there was a large notch in it just to the right of the middle line. At one point just below the ensiform cartilage the liver had an elastic feel, but elsewhere it appeared very firm. The spleen was found to be slightly enlarged. There was no ascites. The urine was normal. An examination of the blood showed that there were 3,400,000 red cells and 6600 white cells per c.mm., and that the hæmoglobin was just under 90 per cent. He was discharged on September the 1st feeling much better, but



Dr. James E. H. Sawyer's Case of Diffuse Angiomatous Condition of the Liver.

Allard & Son, Impr.

with no alteration in the physical signs. He next came up to the hospital on July the 25th, 1905, and was treated in the out-patient department under the care of Dr. Russell for four weeks, but as he made no improvement he was admitted as an in-patient on August the 23rd.

On admission into the hospital the patient was a very small child for his age. His face was very thin and pale, and there was great emaciation. The temperature was 100·2° F., the pulse rate 126, and the respiration rate 36 per minute. The heart appeared normal. There was slight impairment of resonance over the base of the left lung behind, and the breath-sounds were not so clearly heard there as on the right side. The abdomen was very large, and the circumference at the level of the umbilicus was 29 inches. The skin over the abdomen was tense, the veins dilated, and the umbilicus prominent. The liver could be felt extending about two inches below the costal margin in the right nipple line. The surface felt hard and nodular. The spleen was greatly enlarged and felt very firm. There was a considerable amount of ascites. The child passed very little urine, varying in quantity from 4 to 9 ounces in the twenty-four hours. It never contained any albumin. The bowels were opened two or three times a day on the average; there was no diarrhœa. There was no œdema of the legs.

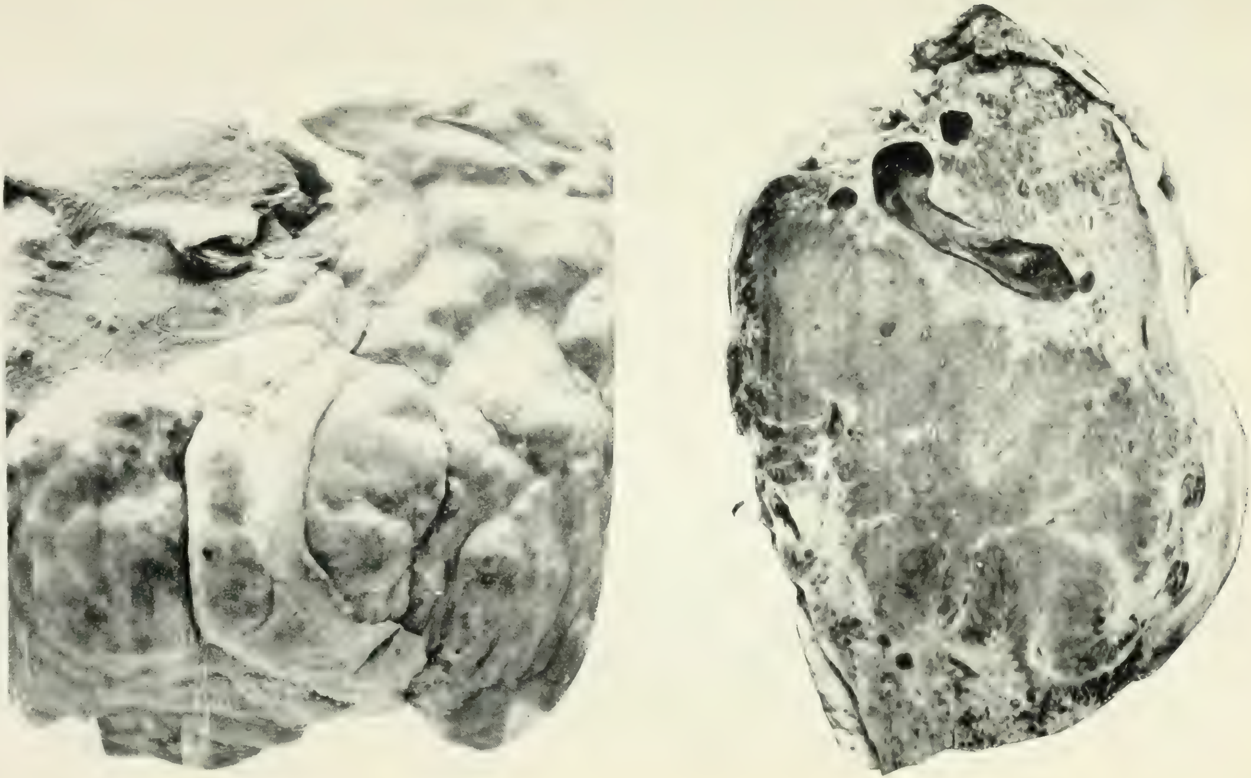
On August the 24th 168 ounces of clear ascitic fluid, with a specific gravity of 1008, were drawn off; but it very rapidly accumulated, and on September the 4th another 100 ounces were removed. The drugs exhibited were liquor hydrargyri perchloridi and potassium iodide. The patient gradually became worse, and coma supervening, death took place on September the 13th, twenty-four days after admission.

The post-mortem examination was made by me twenty-four hours after death. The body was extremely emaciated and very small for the age of the boy. There were no signs of congenital syphilis. The heart weighed 2 oz. There was a slight excess of the pericardial fluid. The right ventricle was dilated and contained blood-clot. There was no endocarditis, and the cardiac muscle appeared normal. There were no abnormal changes in the aorta, and no thickening of any of the arteries of the body. There was an acute hæmorrhagic pleurisy on the left side, which affected the whole of the pleura. There was a little blood in the pleural cavity, and the pleura was thickened, dark red in colour, and had a spongy appearance. Microscopical examination showed this to be an acute inflammatory process, probably tubercular in origin. The right pleura was normal. The lungs were œdematous at their bases, but were otherwise normal. The glands at the root of the left lung showed small foci of caseation. The larynx and trachea were normal. The veins at the lower end of the œsophagus were dilated.

The peritoneal cavity contained 3½ pints of clear straw-coloured fluid. It had a specific gravity of 1013, and contained 0·5 per cent. of albumin. The peritoneum was considerably thickened all over but there were no adhesions. The veins in the stomach showed some dilatation. The small and large intestines were normal and there was no ulceration. The mesenteric glands were slightly enlarged and in one there was a calcareous nodule.

The liver weighed 29½ oz. It was very irregular in shape and there were deep depressions on its surface. It was nodular, and the raised portions, varying in size from that of a small pea to that of a walnut, were of a purplish colour, while the depressed portions were yellow. The capsule was considerably thickened, and especially in certain areas. On section of the liver there were seen to be broad bands of fibrous tissue intersecting what appeared to be the true liver-substance. This was dark-red in colour and much softer than the fibrous bands. The portal veins were considerably distended. The gall-bladder was normal.

The tail of the pancreas was swollen and contained small caseating nodules. The kidneys weighed—right 3 oz., left 3¼ oz. They appeared rather large, but otherwise they looked healthy. Each suprarenal body contained three or



Dr. James E. H. Sawyer's Case of Diffuse Angiomatous Condition of the Liver.

four small caseating points about the size of a pin's head. The spleen was large and weighed $5\frac{1}{2}$ oz. The capsule was considerably thickened and the substance of the spleen was very firm. There were a few small caseating areas. The brain weighed 48 oz. and was normal.

Microscopical examination.—The caseating areas in the spleen, pancreas, and suprarenal bodies were found to be tubercular.

The condition of the liver is very peculiar. There is an extreme degree of cirrhosis, which apparently is of long duration. Broad bands of fibrous tissue intersect the liver and prolongations from these surround the lobules, as in multilobular cirrhosis. In many places at the periphery of the lobules there can be seen a large new formation of bile-ducts. Throughout the liver there is seen what appears to be a diffuse angiomatous condition. It almost looks as though there was an increase in the number of capillaries which have invaded the liver-tissue. These capillaries are greatly dilated, and this dilatation is so extreme in some places that it gives the appearance of a few liver-cells scattered throughout an angioma. In some places the liver-cells are atrophied, and in others they show fatty degeneration, and from other places they have completely disappeared. A few areas of caseation can be made out in the liver, and round these giant cells are seen.

The kidneys show a great dilatation of the capillaries, and this change is chiefly found in the medulla. There are no interstitial changes in the kidneys. The spleen is extremely engorged with blood, and there is a marked degree of fibrosis.

That such a dilatation of capillaries with destruction of the liver-cells could be produced by the cirrhosis appears to be almost impossible. The changes in the kidneys seem to be of a similar nature to those in the liver, but in a much less marked degree. The fact that there is no new formation of fibrous tissue in the kidneys favours the view that the angiomatous condition of the liver is not secondary to cirrhosis. It might be thought that the angiomatous changes in the liver were congenital in origin and the cirrhosis a secondary condition. If this view were accepted the disease would have to be called a diffuse angioma, which would imply that there was a new formation of capillaries, and would explain the changes in the hepatic cells as a result of compression by these vessels. Another explanation for the condition is that it is primarily due to some change in the liver-cells themselves, that some poison has been acting upon these cells, causing their gradual destruction, and that the dilatation of the capillaries is dependent upon the atrophy of the hepatic cells. The same poison might also produce the cirrhosis. (*Exhibited October the* 20*th*, 1905.)

Discussion.

Dr. Theodore Fisher asked what was the condition of the hepatic veins. Obliteration of hepatic veins in rare instances produced a similar dilatation of the vessels of the liver.

Mr. L. S. Dudgeon said if the primary cause of the changes in the liver described by Dr. Sawyer in his case was atrophy of the liver-cells it would be interesting to know why one did not see the same condition in toxæmias, acute yellow atrophy, and in some chronic affections of the liver.

Dr. William Ewart asked whether the dilatation which was shown on one of the slides was due to the section having been made close to the point of bifurcation of some bile-duct. Any dilatation of the bile-ducts must have been due to pressure, and the same pressure might perhaps have had something to do with the dilatation of the blood-vessels.

Dr. E. H. Sawyer (Birmingham), in reply, said he had no other explanation to offer for the condition of the liver than that it was primarily due to some changes in the liver-cells themselves. If that were so the cause must have been going on for a long time, whereas in acute yellow atrophy it was an acute change. He did not detect any dilatation of the bile-ducts.

9. A CASE OF DERMOID TUMOUR OF THE MEDIASTINUM IN A CHILD OF TWO YEARS.*

By GEORGE CARPENTER, M.D.

Ruby T—, aged 2 years, was admitted into the North-Eastern Hospital for Children, under Dr. George Carpenter's care, on June the 22nd, 1905. The previous November she had pneumonia. She was healthy until the preceding July, when she became languid, contracted a cough, and subsequently developed the attack of pneumonia. At this time her chest was noticed to be bulging. Two days before she was taken to the hospital it was noticed that she could not lie down, her respirations were accelerated, and she complained of pain in her side.

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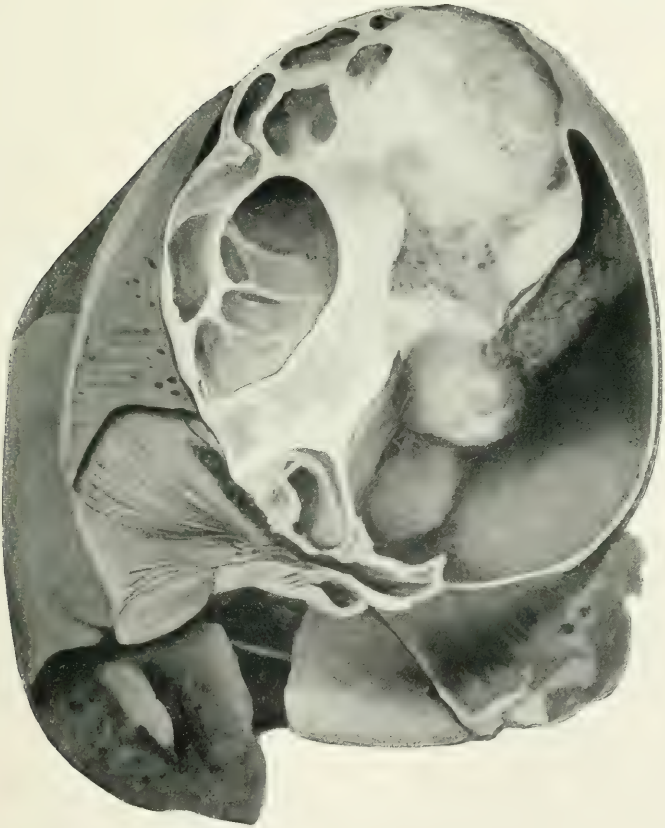
* See 'Lancet,' February the 3rd, 1906, p. 295.

When admitted into the hospital she was cyanosed, there was sucking in of the lower intercostal spaces, mostly on the left side. The respiration rate was 60 to the minute, the pulse rate 136 to the minute, and the temperature 99° F. Her ribs were beaded and the anterior fontanelle was patent. The right side of the chest in front was dull from apex to base; over the apex the breathing was tubular in character, and elsewhere there was deficient entry of air passing on to absent breath-sounds at the extreme base. On the left side in front there was dulness and tubular breathing above and below the clavicle. On the right side behind the chest was absolutely dull; the breath-sounds were bronchial above the spine of the scapula and elsewhere vesicular and diminished, but nowhere absent, not even at the extreme base. Over the left apex there was dulness and tubular breathing. The heart's sounds were best heard over the fifth rib some little distance outside the nipple line, and accompanying the first sound was a systolic bruit. The liver was displaced; its free edge was felt four fingers' breadth below the costal margin in the nipple line, and there was a distinct depression between the liver and the costal margin. Such were the physical signs gleaned at a hasty examination, and aspiration was proceeded with at once in sequence to an exploratory puncture; 13½ ounces of pale, straw-coloured, opalescent fluid were withdrawn. After aspiration the chest became resonant behind. Air entered quite well all over, accompanied by fine râles. In front the chest remained absolutely dull, with deficient entry of air. She was again explored just outside the right nipple and the syringe became charged with blood. There was some hæmoptysis, a few drops after the exploration, perhaps half a drachm of blood. The heart's sounds were now heard loudest just outside the nipple and the organ had probably shifted half an inch or so towards the mid-line; the bruit had disappeared. The liver receded about a finger's breadth. On closer examination it was noticed that the front of the chest was much bulged, it was rounded in outline, and occupied more of the right side than the left. An X-ray examination was made and it was reported by Mr. Sale-Barker that the whole of the right lung was opaque to the rays. The breath sounds on the left side became vesicular. After the aspiration the child rapidly improved. When she left the hospital on July the 3rd the chest was still dull in front on the right side and the cardiac impulse was in the fifth interspace.

On July the 31st she was brought to the out-patient department cyanosed and moribund. She was readmitted into hospital and died shortly afterwards. At this time, on a hurried examination, it was found that the right chest was completely dull, back and front, and the free edge of the liver was four fingers' breadth below the costal margin in the nipple-line.

A post-mortem examination was made the same day. The left pleura was healthy, as also the right pleura. In front of the right lung was a mass the size of the fist. On incising this, from 6 to 8 ounces of clear fluid escaped; inside the cyst was some cheesy material, with a few hairs lying at the bottom. The rest of the tumour felt hard. The right lung was much compressed. Dr. Theodore Fisher, who examined the specimen, made the following report: "The tumour is roughly spherical. In addition to displacing the heart slightly to the left, it projected into the anterior mediastinum and reached to the inner surface of the left lung. The right lung was compressed into an airless strip which lay behind; the compressed lung did not occupy more than half of the space of the lung on the opposite side. On section the tumour is chiefly composed of a considerable number of cysts, the largest of which is the cyst



Dr. George Carpenter's Case of Dermoid Tumour of the Mediastinum in a Child of two years.

Adlard & Son, Impr.

which has been opened. Between the cysts, running from above downwards, is a partition composed of fat and fibrous tissue and containing a few calcareous masses (*vide* Plate III). Most of the cysts contained a gelatinous material of a light-brownish colour. In the large cyst, which had been previously opened, were sebaceous-looking material and short hairs."

(*Exhibited October the 20th,* 1905.)

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10. TWO CASES OF CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By GEORGE CARPENTER, M.D.

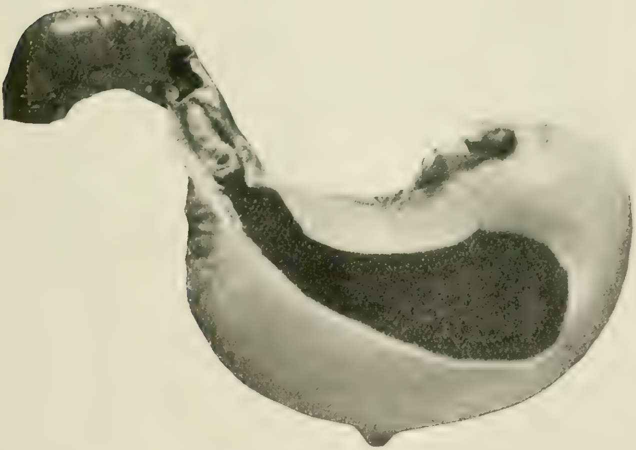
Edward J—, aged 3 weeks, was sent by Dr. Douglas Wright to Dr. George Carpenter, and was admitted into the North-Eastern Hospital for Children on June the 8th, 1905. The child was full term and the sixth pregnancy. There had been neither miscarriages nor stillbirths. The weight at birth was 8¾ lbs. It started vomiting on June the 1st, when a fortnight old; since then it had been sick after almost every feed. Occasionally three feeds were passed by without sickness, but then it vomited a large quantity. Specks of blood were noticed in the vomit, but the mother had a sore nipple. At times the vomit was brown like beef-tea. The bowels were natural until the commencement of the vomiting, when they became costive. Soap enemata were followed by a small, bright green stool. The last motion was sage-green in colour, and of a uniform tarry consistency.

On admission.—A hard tumour, the size of the terminal phalanx of the little finger, was found deep down in the abdominal cavity, above and to the left of the umbilicus. The infant was weighed on June the 12th, and found to be 6½ lbs., a loss of 2¼ lbs. since its birth. Since its admission it had vomited from five to eight times in the twenty-four hours. It was given a mixture of milk and barley-water by the mouth, 1¼ ounces at a time.

June the 15th.—Since admission the bowels have acted irregularly, on three days once or twice in the twenty-four hours, and of a dark-green colour, at others not at all. Vomiting as before. The temperature has been subnormal.

On June the 17th visible peristalsis was first noticed. The child's eyes looked sunken, and it was obviously wasting. The temperature ran up to 101·2° F. Nutrient enemata were ordered. The stools were a little more frequent, the sickness as before. On June the 19th Mr. Ewen Stabb saw the patient with Dr. Carpenter, and it was decided that the pylorus, which felt as hard as iron, should be freely dilated. The stomach was opened by a longitudinal incision. The stricture would just admit a No. 4 gum-elastic catheter. The pylorus was dilated by a pair of blunt-ended dressing forceps. When the dilating was finished the little finger could be passed through the pylorus with ease. A soft catheter was pushed into the duodenum, and a feed of milk and brandy administered. The stomach was sewn up with Lembert's sutures. Owing to the stony hardness of the tumour pyloroplasty was thought impossible of performance, and the simplest operation selected in view of the child's critical condition. The child was collapsed afterwards. The temperature at this time was normal. On June the 20th mouth-feeding was stopped owing to sickness, which occurred after and between the feeds. Albumen-water was given by the mouth, a teaspoonful every half-hour. June the 21st: Albumen-water and about half the rectal feeds retained. Temperature raised since the operation, this evening to 102° F. June the 22nd: Wound dressed. The infant is becoming more wasted and the eyes more sunken. Taking milk and barley-water by the mouth. Since the operation has vomited from two to four times in the twenty-four hours, and the bowels have been opened from two to four times. Temperature 103·5° F. June the 27th: He had lost $1\frac{1}{4}$ lbs. in weight. Vomiting occurred but once on this date, once on the 26th, not at all on the 25th, but four or five times on the 23rd and the 24th. The bowels were opened from three to five times in the twenty-four hours, loose and black stools. The temperature was about 100° F. Mr. Sydney Stephenson was asked to examine the infant's eyes, and reported that there was a septic ulcer taking up two thirds of the right cornea, with protrusion of Descemet's membrane. On July the 2nd the ulcer perforated, and the lens was found under the upper lid. In the meanwhile the child had been sick from two to three times in the twenty-four hours, and the bowels had been opened from four to eleven times. The temperature had been subnormal with one or two exceptions, when it rose to 100° F. He died on July the 4th.

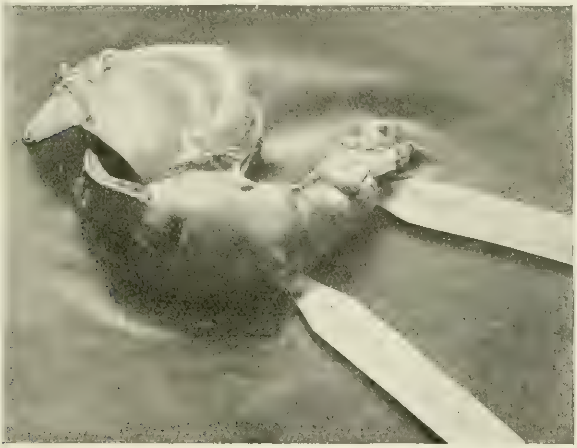
FIG. 1.



The post-mortem examination was made on July the 5th. The wound had not healed. The infant was greatly emaciated. All the organs were normal. The pylorus was as described on physical examination. On filling the stomach with water, none passed through the pyloric opening (*vide* Fig. 1). The stomach held 4 ounces; the whole of the intestines were collapsed. The right eye was completely disorganised from suppuration. The child evidently died of starvation.

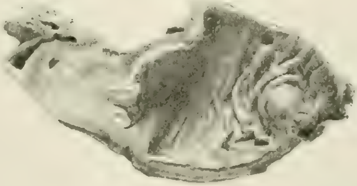
Case 2.—Annie D—, aged 5 weeks, the first infant, was a fine child at birth. She was fed on the breast, and had occasional attacks of sickness. When a fortnight old she was put on the bottle. When three weeks old she had wasted "a good deal." Proprietary foods were then tried and later sterilised milk, but nothing seemed to suit her. By the fourth week she was much worse and either vomited every feed or accumulated feeds in the stomach and then "pumped them on the floor." She was always constipated. She was admitted into the North-Eastern Hospital on July the 10th, and looked somewhat wasted, but she weighed 7¼ lbs. A lump was felt in the region of the pylorus. Under an anæsthetic, which the child took badly, the lump was found to be of cartilaginous hardness and it was grooved transversely. It was thought to be about the size of the terminal phalanx of the index finger, and the stomach walls in its neighbourhood also felt thickened. The intestine (duodenum) also was thought to be somewhat thickened. After the examination the infant was fed by nutrient enemata. Water was given by the mouth, and 10 minims of brandy hourly as it had a very poor pulse. It was sick four times. On the morning of July the 11th, just before the operation, the pulse was weak and the child was somewhat cold. A hypodermic injection of brandy was given. Ether was administered. Mr. Lockhart Mummery operated, and the appearance of the pylorus tallied with the description already given. The growth was of cartilaginous hardness, and the thickening extended chiefly along the greater curvature of the stomach but also along the lesser. Mr. Mummery deemed it impossible to perform pyloroplasty and anterior gastro-enterostomy was done, the operation taking twenty-five minutes to perform. The infant was returned from the theatre in a collapsed state, and very soon afterwards it started continuous vomiting of blood, not in any great quantity at a time, but it lost a good deal in small amounts. A solution of adrenalin (adrenalin solution 1 drm., normal saline solution 5 drms., standing on ice) 10 minims every hour by the mouth stopped the bleeding. The child was fed by the rectum. In the afternoon the temperature began to rise rapidly, and the child became worse. Stimulants were withheld, and morphine gr. $\frac{1}{200}$ was given hypodermically. The pulse became more and more feeble and the child died at 6 p.m., the temperature having risen to 103·8° F.

FIG. 2.



At the autopsy the body was somewhat wasted. The organs were normal. A section of the pylorus measured half an inch across, the increased thickness to the naked eye appearing to be almost entirely muscular fibres. There was also considerable hypertrophy of the muscular wall of the stomach over the pyloric third (*vide* Figs. 2 and 3).

FIG. 3.



The pathological specimens were handed to Dr. P. L. Hipsley for further observations, and he reported as follows: In Case 1 there was nothing unusual about the shape or size of the stomach. The capacity in moderate distension was found to be 4 ounces. The wall was thicker than normal throughout, but the thickening was most marked in the pyloric fourth. The pylorus itself formed a tumour $\frac{3}{4}$th inch long and about $\frac{1}{2}$ inch thick, being definitely marked off from the duodenum but tapering away gradually into the stomach wall. On opening the stomach along the anterior wall midway between the greater and lesser curvatures and continuing the opening through the pylorus the wall of the latter was seen to be about $\frac{1}{4}$th inch thick, and at its junction with the stomach wall it tapered down to about $\frac{1}{8}$th inch. A No. 4 catheter could be passed through the sphincter. The portion of the sphincter projecting into the duodenum looked remarkably like the cervix uteri.

Microscopical examination.—On microscopical examination the principal feature to be seen was the hypertrophy of both the circular and longitudinal layers of muscular fibres. The former was five or six times its normal thickness, and the latter was also hypertrophied but not to the same degree, being perhaps two or three times its usual thickness. The distinction between longitudinal and circular layers was not so sharply defined as in the normal condition. Here and there towards the inner portion of the longitudinal layer bundles of circular fibres could be seen, and in places, moreover, bundles of fibres from the longitudinal layer penetrated the circular and traversed it in different directions and for variable distances. The muscular bundles of both layers appeared to be very densely packed, and there was an increase in the amount of fibrous tissue which is usually seen traversing the circular layer. These fibrous bands were very well demonstrated by staining with the van Gieson mixture, which stains fibrous tissue pink and muscular tissue a yellowish colour. The mucous membrane appeared to be natural, but the submucous layer was thicker than normal and the muscularis mucosæ was distinctly hypertrophied. Very little round-cell infiltration could be seen in either submucous or muscular coats. There were no vascular changes to be seen.

The stomach of Case 2 presented features very similar to those described in the first case. There was no marked dilatation, if any at all, although, unfortunately, the actual capacity of the organ was not estimated. The stomach wall was very much thickened in the pyloric fourth, much more so than in the first case. The pylorus formed a tumour about the size of the last joint of the ring finger. A microscopical examination of both the thickened stomach wall and pylorus was made. The latter presented features identical with those described above. In the stomach wall three layers of muscle could be seen. The inner circular layer was about equal to the thickness of the other two combined. All three layers were hypertrophied. The mucous membrane presented nothing noteworthy, but the submucosa was undoubtedly thicker than normal.

Remarks.—Both these cases in respect to the cartilaginous hardness of the pyloric tumour and to the hypertrophy of the stomach walls resembled a case recorded by myself and Mr. Lockhart Mummery in Vol. V of ‘Reports of the Society for the Study of Disease in Children,’ pp. 242–245, two drawings being given.* This patient, aged 3 months, was operated upon by pyloroplasty but died the following morning. The post mortem showed that a free opening had been established through the pylorus and mechanically the operation was a success. It is not always easy to feel a hypertrophied pylorus, for the tumour is often deep down in the abdominal cavity and is apt to be missed on palpation, but by methodically squeezing the abdominal contents between the fingers and thumb the condition of the underlying viscera can be readily ascertained. This method of examining infantile abdomens, which is a very delicate one and yields admirable results, should always be practised as a matter of routine.†

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* ‘The British Journal of Children's Diseases,’ vol. ii, p. 408.

† *Ibid.*, pp. 247–249, remarks by the author.

Loreta's operation was not attended by success in Case 1. The infant recovered from the shock of the operation, but stenosis recurred and starvation from mechanical causes followed.

Case 2 did not survive the shock of operation, which took the same time to perform, viz. twenty-five minutes, as the case of pyloroplasty previously mentioned. The condition of the pylorus was much the same as in that case, and if pyloroplasty had been attempted the same result would have doubtless followed. In neither case was the physical condition of the infant good, but regarded in the light of cases of marasmus, they had by no means reached that condition of body which is recognised as past hope of recovery. My thanks are due to the house-physician, Mr. E. D. Wolf, for the photographs of the pathological specimens and also for the careful notes taken by him.

(*Exhibited October the 20th,* 1905.)

11. THREE CASES OF CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By EDMUND CAUTLEY, M.D.

I have brought these three specimens before the Society partly to illustrate the condition of the pylorus shortly after the operation of pyloroplasty, partly to indicate the causes of death after operation, and partly because I think in the present state of our knowledge unfavourable results should be noted as well as the satisfactory cases.

Summary.

No.	Sex.	Age at operation.	Weight at operation.	Date of death after operation.	Cause of death.
1	M.	29 days	Under 5 lb. 9 oz.	54th day	Ileo-colitis.
2	M.	53 days	Under 6 lb. 3 oz.	4th day	Syncope.
3	M.	61 days	Under 8 lb.	5th hour	Collapse.

All the cases were hospital patients. This is an important point, for the prognosis is much worse in hospital than in private cases, for the reason that they are usually much more wasted and in a bad state for standing operative measures. The condition has generally been allowed to continue for an unduly long time before the child is brought for treatment. The first case was, however, sent to me early by Mr. P. L. Blaber, of Brondesbury, who had recognised the probability of the affection. The child was an unsatisfactory one, being somewhat premature and very small and wasted. Yet in spite of these defects he lived a considerable time, and only succumbed to an attack of ileocolitis, to which these marasmic infants are peculiarly liable after operation. I am inclined to think that I allowed a too liberal diet in the early stages. This is indicated by the occurrence of the temporary œdema which he developed. Such an œdema is not uncommon in these marasmic infants after operation, and is a very grave prognostic indication. It shows a profound degree of marasmus and inability to assimilate food properly. It was a disappointing case, as at one time the child had gained a pound in weight and seemed well on the road to recovery.

The second case was still more disappointing, for the child appeared to be going on splendidly. He was in the best state of the three for operation, but had rapidly lost weight in the hospital before operation.

The third was the largest child, and yet was extremely wasted and looked much the most ill of the three.

Another point worthy of notice is that one case (No. 3) had been entirely breast-fed. It would, perhaps, be more easy to believe in the spasm theory of causation if these cases only occurred in the bottle-fed. It is difficult to believe that normal human milk can set up such a spasm.

All these children were boys. Probably both sexes are about equally affected, but it is curious that out of the fourteen babies which have come under my notice only four have been girls.

Strictly speaking, it would be justifiable to claim the first case as a successful result as regards the operation. The child's death had nothing to do with the operative measures.

In November, 1903, I reported to this Society (*vide* 'Reports,' vol. iv, 1904) a case with somewhat unusual features. For various reasons I regarded the obstruction as milder than usual and postponed operation, but the boy died at the age of fourteen weeks. Since then I have had two private cases operated on. Mr. C. T. Dent performed pyloroplasty in each, on the thirty-fifth and twenty-ninth day of life respectively. Both infants were girls, fairly well nourished, and weighed 7 lbs. 6 oz. before operation. Both recovered without bad symptoms, and are now strong, healthy children. Thus, out of my last six cases three fatal ones were hospital patients, two private patients recovered, and the sixth was a hospital patient who died unoperated upon. I draw attention to these facts because it is sometimes stated that operation is not necessary, and that patients will recover without. It may be so in a few instances. My own experience is that death is the common sequel if no operation is done.

The fourth case is different in character and is recorded to illustrate the difficulty of diagnosis, the risk of submitting a babe to an unnecessary operation, the possibility of diagnosing hypertrophic stenosis of the pylorus erroneously, and the possibility of curing such cases by lavage and other measures, and then concluding that operation is not necessary for the cure of pyloric hypertrophy. The case is also of very great value in favour of the view that continued vomiting does not give rise to pyloric hypertrophy, even though it be ascribed to pyloric spasm.

(1) Richard H— was born on November the 4th, 1904, three weeks prematurely. He appeared healthy and was thought to weigh about 7 lbs. He was bottle-fed for two weeks and did fairly well but was occasionally slightly sick. The vomiting became more persistent and after the 26th almost constant. It varied in character, food being sometimes kept down for an hour or two and then ejected

violently, sometimes rejected at once. The motions were always small, dark-green, and contained no curds.

On admission into the Belgrave Hospital on December the 1st he weighed 5 lbs. 9 oz. The stomach was not dilated but the pylorus could be telt. Next day peristalsis was noted. The child sometimes took well, seemed ravenous, but was soon satisfied, not taking more than an ounce at a time. The vomit was sometimes ejected violently a long way, even as far as 3 ft. The stools were greenish-brown, small, rather dry, and contained no fæcal matter.

Pyloroplasty was done by Mr. C. T. Dent on December the 3rd. The condition was typical and the single mucous fold, resembling a verumontanum, was marked; the operation took twenty-four minutes.

Subsequently the child progressed as favourably as could be expected when its marasmic state was considered. Moderate fever lasted for three days. Vomiting ceased fifty-five hours after the operation. On December the 16th he weighed 6 lbs. 9 oz., and was going on well except that the stools were not quite normal and his progress had been temporarily delayed by œdema. On December the 29th he had an attack of ileo-colitis, from which he recovered; but, unfortunately, he had another attack on January the 23rd and died two days later. The stomach was found partially adherent to the liver, and the pyloric canal was amply large enough. The child recovered from the pyloric condition but succumbed to marasmus and ileo-colitis.

(2) Leslie George E—, was born on June the 11th, 1905, at full-time. He was the sixth child and a fine baby. His food consisted of milk and barley-water. During the first week of life vomiting was almost incessant. Nothing except meconium was passed by the bowel. In the second and third weeks the vomiting was less frequent. In the fourth week he vomited occasionally and normal stools were passed. In the fifth and sixth weeks the vomiting became very frequent and constipation was marked, the bowels not acting for a fortnight.

When admitted into the Belgrave Hospital on July the 27th he weighed 6 lbs. 12 oz., was much wasted, vomited his food, and passed small green stools. Up to August the 1st the vomiting remained about the same. He had lost 9 oz. in weight. His bowels acted daily and contained curds. On this day the pylorus was distinctly felt as a hard, movable lump, the size of a small marble, somewhat like a gland. Peristalsis was visible.

On August the 2nd pyloroplasty was performed by Mr. Dent. The condition was typical. The operation lasted thirty-five minutes and the child took the anæsthetic well.

Vomiting ceased thirty-nine hours after the operation. On one occasion, thirty-two hours after operation, a large amount of fluid was vomited and the child became collapsed. He revived on the administration of ten drops of brandy and a subcutaneous saline injection of 3 oz. On August the 5th, at 7.0 a.m., the child seemed to be going on splendidly. He was a good colour, had an excellent pulse, and was sleeping quietly. Half an hour later he died quite suddenly. The temperature had never been over 102° F. and had come down to 100° F. two hours before death, and there had been no return of the vomiting.

On post-mortem examination the result of the operation appeared excellent. The pylorus looked quite healthy and the canal was amply patent. The stomach was a little dilated. Digested food was present in the small and large intestine. A little lymph was deposited on the anterior surface of the stomach and along the edge of the right lobe of the liver. The cause of death is not quite clear. I have, however, little doubt that it was due to a syncopal attack, similar in character to the one which had occurred two days previously, but more sudden and severe.

(3) John B— was born on May the 25th, 1905, at full-time. He was the third child of healthy parents and weighed over 9 lbs. He was breast-fed until July the 22nd. Vomiting began in the first week of life and continued, becoming more pronounced during the fourth and

fifth weeks. Constipation had been present throughout and the child had steadily wasted.

When admitted into the Belgrave Hospital on July the 24th he weighed 8 lbs. 4 oz., was very pale, much wasted, and had a weak cry. He was given rectal feeds, but passed one dark, semi-solid stool, like black paint, before rectal feeding was begun. Peristalsis was visible and the stomach a little dilated. The abdomen was quite lax, but the pylorus could only be felt very indefinitely, if at all. In spite of cocaine and feeding on teaspoonfuls of whey every quarter of an hour the vomiting continued up to 1 a.m. on the morning of the 26th. After that he retained some whey. The temperature was 103·4° F. on the morning of the 25th, but fell to 99·8° F. on the 26th.

On July the 26th pyloroplasty was done by Mr. Dent and took thirty-five minutes. The child took the anæsthetic badly and became collapsed afterwards. It never properly recovered and died from collapse five hours after operation.

The pylorus presented the typical condition, but was not quite as large as usual. The fold of mucous membrane was very marked indeed.

The boy died because of his marasmic and anæmic state at the time of operation, having lost a further 4 ounces in weight since admission. In this condition his strength was insufficient to withstand the strain of the operation and the effects of the anæsthetic.

(4) Marjorie C— was born on July the 25th, 1905, at full-time, and weighed 7½ lbs. For three days she was breast-fed, but vomited. After that she had milk and barley-water, Allenbury No. 1 food, Horlick's malted milk, and Savory and Moore's food for variable periods, and more recently was put back on a diet of milk and barley-water, with occasional feeds of the Allenbury food. Throughout the vomiting had been persistent and of varying severity, but it was not associated with constipation. Wasting had been more marked during the two weeks before admission. She was sent up to the Belgrave Hospital on October the

13th, as possibly a case of pyloric hypertrophy, by a doctor who had seen one of these cases. On examination she weighed 6 lbs. 1 oz., was markedly wasted, with wrinkled, lax skin, anæmia, depressed fontanelle, and apthæ on the tongue. The abdomen was lax and empty, but the pylorus was not palpable, and there was no peristalsis or dilatation of the stomach. From the absence of these signs and of characteristic projectile vomiting and of constipation, I concluded that the case was not due to pyloric hypertrophy, but was simply one of marasmus from pyloric spasm and erroneous feeding. The child died from exhaustion on the morning after admission. She had had no further vomiting, retained more than 16 ounces of whey, and had passed two stools.

On post-mortem examination no evidence of organic disease was found. The pylorus (shown at the meeting) was easily patent and showed no evidence whatever of hypertrophy. Yet, in this instance, vomiting had begun shortly after birth and persisted up to death—a period of over eleven weeks. Such a case as this, if it ended in recovery, might very easily be mistaken for one of congenital hypertrophic stenosis of the pylorus, cured without recourse to operation. (*Exhibited October the* 20*th*, 1905.)

12. A CASE OF SPINA BIFIDA OCCULTA (?).

By FRANCIS JAFFREY.

Mr. Jaffrey showed a case of spina bifida occulta in a girl aged $1\frac{7}{12}$th years. A swelling was first noticed in the lumbo-sacral region when the child was eight days old. In the centre of the swelling there is a small depression, and the swelling itself appears to be composed of fat; it has not increased in size, there is no paralysis of the lower extremities, but there is marked enlargement of the stomach. As it does not inconvenience the child no operative treatment is proposed.

(*Exhibited November the* 18*th*, 1905.)

Discussion.

The Chairman (**Mr. R. Clement Lucas**) explained that Mr. Jaffrey was inclined to withdraw the word "occulta," as the nature of the case seemed fairly evident. He asked what Mr. Jaffrey proposed to do in the way of cure.

Mr. Thomson Walker said it seemed to him that the vertebræ were closed and that there was a fatty tumour over the closed vertebræ. In cutting sections of spina bifida the cord was frequently spread out, and in the open part of the cord one found a fatty tumour. It was possible that this case represented a similar condition in which the vertebræ had closed and the fatty tumour was excluded from the spinal canal. In answer to the Chairman (Mr. R. Clement Lucas) he could not discover any deficiency in the vertebræ, but it was not easy to feel through a fatty tumour.

Mr. Francis Jaffrey, in reply, said he did not propose to treat the case at all, because the child had no bad effects. He thought he could feel a small hole between the last lumbar and the first sacral vertebræ. Mr. Bland-Sutton, in his book, described and illustrated a case of spina bifida in which there was fat in the centre and fat all round. But in that case it was pedunculated. He agreed with Mr. Thomson Walker that it was mostly composed of fat in his case.

13. A CASE OF DOUBLE HARE-LIP AND CLEFT PALATE.

By G. R. WILSON, M.B.

Mr. Wilson asked for opinions as to the best time for operating on such conditions and the method which should be carried out. The age of the infant was 1 month.

(*Exhibited November the* 18*th*, 1905.)

Discussion.

The Chairman (**Mr. R. Clement Lucas**) said it was a moot point as to what was the best age for treating hare-lip and cleft palate. Certain surgeons thought it was wise to begin at once, at a very early date, and operate on the cleft palate before attacking the hare-lip, whereas many, among whom was Mr. Berry, who read a paper on the subject before the British Medical Association at their last meeting, giving reports of cases, held that the old view was the sound one—that it was better to cure the hare-lip first, by which means there was brought about contraction of the palate. It was asserted that those cases which were operated upon early were successes for the moment, and the child swallowed better afterwards, but when it came to the talking period they were not such successes as those which were operated upon at three years of age. He would be very glad to hear the opinions of members on those questions.

Mr. H. S. Clogg thought that very few of the cases of the degree shown ever presented themselves again at the third year to have the operation done. The cases which came back for operation were those with medium-sized clefts. If the patients with wide clefts did not come for operation what became of them? He thought they died early, probably through some neglect in the matter of feeding. Mr. Berry, in his paper, probably mentioned the right age for many cases, but he (Mr. Clogg) thought that children with extensive clefts should be operated upon early. With regard to the shock of the operation, he had had the opportunity of seeing many cleft palates in infants operated upon, and had operated upon some himself, but he never remembered seeing any particular shock as the result.

The Chairman (**Mr. R. Clement Lucas**), in further remarking upon the case, said it was his belief that the children born with defective palates and hare-lips were not favourites with their parents, who often desired that they should not live. He had ceased operating upon out-patients for hare-lip because he saw that the child was afterwards systematically neglected, and that the decline in health and death might be attributed to the operation. Yet among the cases taken into hospital and kept there for a time there was a very small mortality. In the case brought forward he noticed it was possible to push the two sides together, and the cleft could be much reduced in this way. Therefore one could understand that the cleft would have a tendency to diminish if the hare-lip were first operated on. A number of cases were now operated upon by the so-called "flap" method, a procedure which his colleague Mr. Davies-Colley first proposed and which had been modified by Mr. Arbuthnot Lane. But the flap method only provided for the hard palate, and it was maintained by those who opposed the operation that the soft palate was the essential part for speaking, and that it could only be well joined later, and that the result of the early flap operation was not satisfactory so far as speaking went. The number of those operations which had been done on infants was not great, and perhaps there had not yet been time to decide which was the best operation.

14. DEFORMITY OF THE HAND FOLLOWING THE USE OF SPLINTS.

By P. LOCKHART MUMMERY, B.C.

The patient, a boy aged 12 years, was treated for an injury to the wrist at a hospital in London by the application of splints. Ulceration of the skin on the anterior and posterior aspects of the limb followed and the contraction of the wrist and fingers subsequently occurred.

The right hand is much contracted. The fingers are flexed into the palm of the hand, and the wrist is flexed on the forearm to nearly a right angle. There is much wasting of the hand and forearm muscles. There are extensive scars on the back and front of the forearm.

There is slight movement in the fingers and also at the wrist, which seems to indicate that there is no real muscular paralysis. Tactile sensation is unimpaired in the fingers. This condition has been called Volkmann's contraction.

Mr. Dudgeon in 1902 wrote an interesting paper on this condition, with a description of fifteen cases.

Mr. Rowlands recently published a case in the 'Lancet' which he treated by operation and reviewed the previous literature. In nearly all the cases there is a history of splints having been applied too tightly to an injured wrist, and in a large number of the cases sloughing has occurred from the pressure. This has occurred in my case.

The condition is not due to paralysis or nerve injury; as a rule no real paralysis exists and sensation is unimpaired. The condition is the result of inflammation in muscles and contiguous strictures, though whether produced by prolonged anæmia of the muscles or by direct injury is doubtful. Injury of the muscles by too tight splinting combined with the initial injury seems to be the most probable cause; the subsequent formation of adhesions within and between the muscles and in the tendon sheaths with secondary contraction is doubtless the cause of the contraction.

The chief reasons why the condition is always a flexor one are probably that the injured hand naturally tends to fall into the flexor position when not used owing to the weight of the hand, and also that the flexor muscles are the most powerful.

The best treatment for severe cases when the contraction has existed for a considerable time would seem to be shortening the ulna and radius by resection. This operation was first recommended by Mr. Raymond Johnson. Mr. Rowland's recently reported case is most encouraging for this method of treatment. In less severe and more recent cases a prolonged course of massage and exercises is often successful.

(*Exhibited November the* 18*th,* 1905.)

Discussion.

The Chairman (Mr. R. Clement Lucas) remarked that those cases were amongst the most distressing ones to be met with, because they invariably reflected upon the treatment which had been adopted, the splints having been bound on too tightly by the practitioner. But for treatment afterwards they were equally distressing. He could not say that he had had anything but very modified successes with them. The new suggestion of shortening the bones seemed to offer some future prospect of relief. But he did not quite agree with all Mr. Mummery said on the subject, because when the splints were taken off the hand was found to be completely paralysed, showing that the nerves were seriously damaged in the early stages by the pressure, and that the condition was not entirely due to the myositis and inflammation of cellular tissue and skin as the result of the pressure. The hand would generally recover in part, unless the nerves were actually destroyed. He would be glad if Mr. Mummery would bring the case again before the Society after he had operated upon it.

Mr. Lockhart Mummery, in reply, said he would show the case to the Society again, whatever the result.

15. TWO CASES OF RINGWORM OF THE SCALP TREATED BY THE ONE APPLICATION OF THE X RAYS.

BY GEORGE PERNET.

CASE 1.—Cured by one application of the X rays. A girl, aged 9 years, with a patch of tinea tonsurans (*Microsporon Audouini* variety) about 1½ inches in diameter on the vertex, first seen in July last. On July the 17th the diseased area was exposed for twenty minutes in accordance with the Sabouraud-Noiré method. As the members could see, the hair had grown well, so much so that it was not easy to make out where the patch had been. No diseased stumps had been found. The case was a successful cure.

CASE 2.—A boy, aged 11 years, with a patch on the vertex which was in process of cure after a single exposure of twenty minutes. The stumps had come out very well. The fungus was the Microsporon.

With regard to *Microsporon Audouini*, it was the fungus commonly met with in ringworm of the scalp in this country * and in France. But in Germany this variety was very rarely seen. When we considered the enormous amount of time lost in the aggregate by children of school age, the prevalence of ringworm became of national importance. In this respect we were handicapped *quâ* education as compared with Germany, a point to be borne in mind.

With regard to the action of the X rays, they were not parasiticide, so an ointment had to be used simultaneously and every care taken to prevent the spread of the disease. The stumps and hairs affected by the X rays exhibited a stake or pencil-like pointing of their proximal end which was very characteristic.

(*Exhibited November the* 18*th*, 1905.)

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* Pernet, "One Hundred and Thirty Cases of Ringworm observed in the Skin Department of University College Hospital," 'Lancet,' 1898.

Discussion.

Mr. Milner Burgess (Harlesden) asked whether the cases such as Mr. Pernet had described were followed by permanent baldness after the X-ray treatment.

Mr. George Pernet, in reply, said permanent baldness did not result from the treatment. This was shown by the growth of hair in the case of the girl exhibited that afternoon. Further, it was a fact that it was no easy matter to remove hair completely by the X rays. It required a large number of applications, and there was always the risk of setting up a dermatitis which might prove disfiguring.

16. OBSTRUCTION OF THE CENTRAL ARTERY OF THE RETINA IN A GIRL OF FIFTEEN YEARS.

By SYDNEY STEPHENSON, C.M.

Lilian R—, aged 15 years. First seen on October the 27th, 1905, on account of sudden loss of sight in the left eye.

History of present illness.—Yesterday whilst walking in the street the patient suddenly experienced what she describes as a "dazzling" in her left eye, and found that she could see parts of objects only. This occurred about 8.30 a.m., and by 9 a.m. she could perceive a "speck of light" with the affected eye. There was no pain, headache, vomiting, or giddiness. Nothing of the kind had ever occurred before. Patient thinks that her sight is now slightly better than it was. She states that the night before the attack her legs ached and she felt very tired, but apart from this she is in her usual health and was making no particular effort at the moment the attack came on.

Family history.—Lilian is the eldest of six children, all healthy. No stillbirths or miscarriages. The mother had rheumatic fever twelve months before the birth of the patient.

Personal history.—Bronchitis and pneumonia at eighteen months. Measles at five years, preceded by epistaxis, from which the patient continued to suffer frequently until she was eleven years of age. She then developed "anæmia," became short of breath, and experienced pains in her joints. This condition (almost certainly one of subacute rheumatism) persisted on and off for five months, although it was not severe enough to necessitate confinement to bed. Although the girl had never menstruated, the mother states that a slight, non-coloured discharge had been noticed periodically. No history of chorea.

Present condition.—Pupils equal. The right pupil is active to light, but the left one is almost motionless. On the other hand, the indirect (consensual) response of the left pupil is greater than that of the right. Ophthalmoscopic examination of the left fundus showed that the optic disc was indistinct, veiled, as it were, and that the central part of the retina was very pale, except as regards a small area contiguous to the lower-outer side of the disc, where the normal red was retained. Pressure upon the eyeball elicited no pulsation of the retinal arteries, which were reduced in size, although containing red blood. A small, dark-red spot present at the fovea centralis. The periphery of the fundus showed no departure from the normal. The sight of the eye was reduced to the appreciation of shadows merely. This was due apparently to the retention of a small piece of the visual field corresponding to the retained area of retina on the outer side of the optic disc. It could not be mapped out on the perimeter, but the patient could recognise a sheet of white paper there and nowhere else.

The sight of the right eye was more than normal (V. = $\frac{6}{5}$), and the fundus healthy.

Notes made November the 3rd, 1905.—Patient appears to be in good health. Pulse rate 96, regular. Cardiac dulness extends 1½ fingers' breadth to the right of the sternum in the fourth interspace. No cardiac murmurs or, at least, none actually detected in the noisy out-patient room, where the examination took place. At the same time, the first sound of the heart was what might be termed "murmurish."* Urine—sp. gr. 1024, alkaline, with deposit of phosphates; neither albumin nor sugar. No signs of hereditary syphilis.

Eyes.—Hippus in both eyes. Consensual response of the left pupil to light greater than that of the right. L. V. = $\frac{6}{60}$. A small area of the visual field, near the centre, is retained. The margin of the optic disc can just be distinguished, and is best defined at the lower and outer parts. With the exception of a portion of healthy retina, about the size of the disc, and lying to the outer side of the latter structure, the papilla is surrounded by a considerable area of retina, the colour of which is pale. The macula is represented by a dark red, maroon-coloured spot, lying towards the centre of a more markedly pale portion of the affected area. External to the spot and a little above it is a smaller spot, of light red colour, evidently a hæmorrhage. The periphery of the fundus is normal. The retinal veins are tortuous, and decrease rather than increase in size as they approach the optic disc. They are obscured in parts by retinal œdema and pulsate on pressure. The retinal arteries are somewhat contracted, and pressure causes neither pulsation nor segmentation of their contents. The right eye is normal as regards both sight and condition of the fundus.

Notes made November the 17th, 1905 (twenty-two days after the illness began).—The left optic disc is now clearly defined and very pale. Retinal arteries small but contain red columns of blood. Central region of the fundus is less œdematous than on date of last note. The dark red spot at the macula now shows several small yellowish-white patches, such as may be seen when a hæmorrhage is undergoing metamorphosis. The small ecchymosis mentioned as present on the outer side of the yellow spot region can still be recognised.

Remarks.—Obstruction of the central artery of the retina, irrespective of the cause, is not a common condi-

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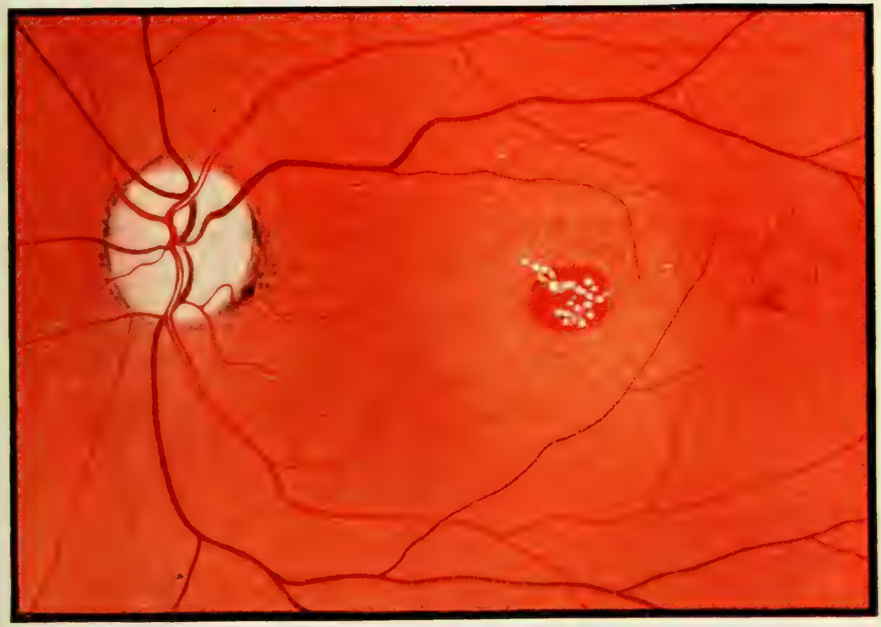
* Several members of the Society who examined the patient's heart at the meeting where the child was shown thought that there was at intervals a faint mitral systolic bruit.—S. S.



Left eye. Erect image.

Mr. Sydney Stephenson's Case of Obstruction of the Central Artery of the Retina in a Girl of Fifteen Years.

Adlard & Son, Imp.

tion under any circumstances. In children it is, without doubt, extremely rare. Personally, the present is the first case I have seen in a patient so young as fifteen years.

The possible causes of the obstruction are four or five in number, *viz.*: (1) embolism; (2) thrombosis; (3) endarteritis obliterans; and (4) arterial spasm. As to the ophthalmoscopic appearances of obstruction being simulated by (5) hæmorrhage into the sheath of the optic nerve, the researches of Gonin ('Annales d'oculistique,' February, 1903) seem to render that contingency something more than merely doubtful, despite the *dicta* of some of the earlier ophthalmologists, notably Magnus. As regards the present case spasm of the central artery may be excluded, since the symptoms have lasted too long to render that explanation probable, unless we assume that the initial spasm has been followed by actual thrombosis, an hypothesis for which in this case there appears to be no ground. Endarteritis would be uncommon in a girl of the patient's age, especially in the absence of hereditary syphilis. In brief, the obstruction is almost certainly due either to embolism or thrombosis of the central artery. In attempting to decide between these two conditions it may be pointed out that no symptoms suggestive of thrombosis can be elicited, such as cerebral accompaniments or a history of former attacks of blindness in the same or the other eye. On the other hand, the history of rheumatism, the existence of a valvular lesion, the sudden nature of the blindness, and the red spot at the macula tell in favour of an actual embolic obstruction of the central artery of the retina.

The so-called "cherry-red spot at the macula," representing the fovea centralis, is a prominent diagnostic feature in recent cases of embolism of the central artery of the retina. It is usually looked upon as a contrast phenomenon—that is to say, as due to the red colour of the choroid showing through the fovea centralis, the thinnest part of the retina. While this is doubtless the explanation in many cases it certainly does not apply to all. The present case is an instance in point, since the red spot, twenty-two days after the beginning of the illness, shows several small, yellowish-white deposits, which can scarcely be due to anything else than retrograde changes in an actual hæmorrhage.

Attention may be directed to the fact that a small portion of the retina, lying in the immediate neighbourhood of the lower temporal quadrant of the optic disc, has retained its normal colour, although the rest of the posterior pole of the fundus presented a milky opacity from retinal œdema. On careful examination at the first visit (October the 27th, 1905), the unaffected part could be seen to be supplied by a cilio-retinal vessel—*i. e.* an off-set from the short posterior ciliary arteries, derived either through the choroidal system or through the anastomosis of Zinn, surrounding the optic papilla. This area, which had about the size of the optic papilla, was the only part of the visual field that retained its functional activity. Cases have been known, indeed, where central vision remained normal, owing to the presence of such a supplying vessel, notwithstanding obstruction of the central artery of the retina. Such a case has recently been published by Dr. Percival J. Hay ('Ophthalmoscope,' June, 1905), where the whole of the macular region derived its blood-supply from a cilio-retinal artery. These tiny vessels, therefore, possess practical importance, more especially as they occur in about 10 per cent. of normal eyes.

The improvement in sight (shadows to $\frac{6}{60}$) that has taken place since the girl was first seen three weeks ago, is probably due to the formation of a collateral circulation through the anastomosis of Zinn. It may, of course, be also due to shrinking of the clot in the artery, assuming that to be the immediate cause of the obstruction.

(*Exhibited November the* 18*th*, 1905.)

Discussion.

Dr. F. J. Poynton asked how frequently the condition described by Mr. Stephenson occurred. He agreed with Mr.

Stephenson that there was some cardiac lesion present, mitral in situation, and that the most likely explanation seemed to be that there was an embolus. He wondered one did not meet oftener with embolism of the retina in rheumatism than was the case. It was particularly interesting as bearing on the question of chorea, for in some cases of chorea after rheumatism the children had onsets as sudden as had those cases of embolism of the central artery of the retina. He remembered the case of a little girl who was walking along the street when she suddenly had a pain in the head and fell down, and when she was taken home in the evening she had well-marked chorea. Other cases of the sort had come under his notice, and it suggested that the same sort of process went on occasionally in cases of chorea. Dr. Kirkes many years ago demonstrated a fairly large embolus in a case of chorea. He asked whether there had been any purpura. Those cases in which a small purpuric spot occurred in rheumatism might be of the same nature.

Mr. Sydney Stephenson, in reply, said he thought obstruction of the central artery of the retina was a very rare occurrence under any conditions. He was not able to quote precise figures, but he had never seen such a case in a child of fifteen years before. With regard to Dr. Poynton's observation on chorea, it derived additional interest from the fact that a certain number of cases of embolism of the central artery of the retina had been reported in children suffering from chorea and in whom no other etiological factor could be found. With regard to purpura, he had not inquired or examined with reference to that, but he would bear it in mind and report as to its occurrence.

17. A CASE OF ATROPHIC PARALYSIS OF THE RIGHT UPPER LIMB (? ANTERIOR POLIOMYELITIS).

By E. FARQUHAR BUZZARD, M.D.

The patient, a girl aged 10 years, is suffering with an atrophic paralysis of the right arm—probably anterior poliomyelitis. The onset was sudden, without injury or illness or pain. At the age of three years a doctor diagnosed dislocation of the shoulder and bandaged the arm to the chest for three months.

There is now atrophic paralysis of the muscles supplied by the seventh and eighth cervical and first dorsal segments of the cord. In addition to the clavicular fibres of the pectoralis major and the posterior fibres of the deltoid are wasted. The pectoralis minor can be easily felt owing to the wasting of the upper pectoralis major fibres. The following muscles are more or less completely atrophied and paralysed, viz. flexors of wrist, pronators, extensors of fingers, and the intrinsic muscles of the hand. There are no oculo-pupillary signs.

(*Exhibited November the* 18*th,* 1905.)

18. REMARKABLE RESULT OF ANTERIOR POLIOMYELITIS.

By F. J. POYNTON, M.D.

The patient is a boy aged $5\frac{1}{12}$th years. Eight weeks ago he returned home from school with a severe headache. He remained awake all that night and screamed a great deal, complaining of pain in his head. Next morning he was drowsy and remained so throughout the next day. That evening his neck and left arm and leg were noticed to be stiff. He was feverish, and apparently had pain and tenderness all over his body. After being in bed for a few days, weakness on the left side was noticed. For three weeks the head was drawn back, and at about the end of this time he got an attack of dyspnœa ; he choked and tried to cough, but was unable to, and his voice changed. He has had no vomiting or blindness during the illness.

Present condition.—When admitted into the Hospital for Sick Children, Great Ormond Street, a week ago, he was in the same condition as now. He has a very widely distributed loss of power. There is very definite weakness of the deltoid and trapezius on the left side and less obvious weakenss on the right. The left triceps is weaker than the right. There is general weakness of the trunk muscles. He cannot hold up his head, and sits stooping forward with a definite lateral spinal curvature, with its convexity to the left in the lower dorsal region. The chest does not expand with inspiration, except by the aid of the accessory muscles, due to intercostal paralysis. He cannot get into the sitting posture without help, due to weakness of the abdominal muscles. The left leg is distinctly weaker than the right. Weakness of the ileo-psoas group does not allow of voluntary flexion of the left hip. Otherwise there is no absolute loss of movement in the legs. The left calf is a quarter of an inch smaller round than the right. He stands with a broad base, and shows definite lordosis. The cranial nerves are unaffected. There is no loss of sensation. The reflexes are everywhere obtained. His mental condition is good.

Electrical reactions.—There is diminished reaction to faradism in the deltoid, trapezius, intercostal, and abdominal muscles, but as the stimulus necessary for contraction causes considerable pain, the reactions have not yet been fully worked out. (*Exhibited November the* 18*th*, 1905.)

19. INFANTILE PARALYSIS OF ABDOMINAL MUSCLES, BOTH LEGS AND ONE ARM IN AN INFANT OF TWO-AND-A-HALF MONTHS.

By GEORGE CARPENTER, M.D.

Eric D—, aged 2½ months, was sent to me by Dr. Cardy Bluck of Southend-on-Sea, and was admitted into the North-Eastern Hospital for Children on October the 5th, 1905. Six weeks before, when being prepared for bed, he commenced to scream and cried throughout the night. He became feverish and refused his food, and three days after the onset "an eye was turned up." He was then "paralysed in all his limbs," and he was thought to be blind, He paid no attention to light when a match was struck. He could not be heard to cry or cough, although faces suggestive of these co-ordinated actions were being made.

On admission he weighed 10 lbs. 12 oz., and there was slight splenic enlargement. He could move his right hand, but the rest of the limb and the shoulder muscles were paralysed. The Electrical Department reported on October the 16th that there was no reaction of degeneration in the arms.

The right foot was not affected, but the rest of the limb was paralysed. The left leg was completely paralysed. In the left leg reaction of degeneration was present. The right leg showed loss of reaction to faradism, but no polar change to galvanism. The abdominal muscles were completely paralysed, and they did not react to faradism.

November the 17th.—Since he has been in hospital he has lost a quarter of a pound in weight, but he can now move the whole of the right arm, though it has but little power.

When he cries the abdomen on the right side, just above the anterior superior spine, shows deep furrowing from the action of the abdominal muscles accompanied by ballooning of the belly wall above it.

On the left side there appears to be some action of the lower abdominal muscle-fibres in a similar situation, and a tendency to grooving, and when he cries this side of the abdomen seems to become a little stiff. On the ballooned side the muscle feels flaccid, but flaccidity is not so marked on the corresponding side. The legs remain as on admission into hospital.

Electrical reactions taken later under an anæsthetic ($CHCl_3$) were as follows:

Right leg.—The hamstrings and all muscles below the knee react well to faradism and normally to galvanism. The extensors of the knee, however, do not react to faradism, but well to galvanism.

Left leg.—None of the muscles respond to faradism, but react well to galvanism.

Abdomen.—The abdominal muscles on both sides react to both currents below a line drawn from the tip of the ninth rib to a spot about half an inch above the umbilicus; above this electrical reactions cannot be obtained to either current. The reaction on the right side, however, is better than that on the left. The rectus abdominis does not react.

Upper limb. Right.—The deltoid, biceps, and pectoralis major do not react at all to faradism but respond feebly to galvanism, the feebleness probably being due to extreme atrophy of the muscles; the forearm muscles react well to both currents.

Left.—Reactions normal to both currents.

(*Exhibited November the* 18*th*, 1905.)

Discussion.

Dr. W. Cardy Bluck (Southend-on-Sea) said that when the child was brought to him he thought it was suffering from tubercular meningitis, as there was a strong history of tuberculosis in the parents, and the children were living and sleeping in the same room. He afterwards changed his opinion. The singular point was that at first the child could be seen to cough and cry, but nothing could be heard.

20. A CASE OF HYDROCEPHALUS IN A BOY OF EIGHT YEARS.

By GEORGE CARPENTER, M.D.

George S—, aged 8 years, under the care of Dr. Cardy Bluck, of Southend, was admitted into the North-Eastern Hospital for Children on November the 10th, 1905. His family history is unimportant. He was a wasted child when born, but his head was not larger than normal. He had measles and whooping-cough at two years old, and about this time, in September, 1899, was a patient at the Great Northern Hospital. At three years of age his mother noticed that his head was large. When four and a half years old, in February, 1902, he was taken to Great Ormond Street Children's Hospital, and was under care there for nine weeks. When he was admitted into the hospital he could walk, but was not able to do so on his discharge. When at Great Ormond Street Children's Hospital he had hydrocephalus and he was thought to have a cerebral tumour, but no localising symptoms were observed. He has had only one attack of convulsions during the last three years, and they were general and not local. Dr. Bluck saw him immediately after his return from Great Ormond Street, and he has been under his care ever since then. He has had at times a certain amount of twitchings, but they were not confined particularly to one side. Eighteen months ago he could just sit up, but not since then. He has also lost control of his water and fæces at times, but there are occasions when he asks. Some years ago he was very slow of speech, but latterly he has been somewhat better. Some days he is better than others. He suffers from headaches at times; three years ago he had a very bad attack, and this seems to have affected him mentally. He is now becoming more helpless, and his head is increasing in size, an inch in three weeks. For the last two weeks there has been a tendency to choke at meals and his breath has been short, but neither have been noticed since admission. His head is now very large, and measures 25½ inches in circumference. From ear to ear it is 16 inches and from the gabella to the external occipital protuberance it measures 18½ inches. In outline it is wedge-shaped with the apex forwards, and it is almost symmetrical. The forehead is pointed, the temporal regions prominent, and the occipital region is flat. The fontanelles and the sutures are closed. The temporal veins are enlarged. The hair is long and curly in front, behind it has been cut short. The right optic disc is bluish-white in colour, and the arteries are small. The optic disc in the left eye is not so full, the arteries are small, and the veins are tortuous. A year previous I examined the eyes at Southend, and there was marked papillitis on both sides. The boy is very fat, and he weighs 4 stone. His speech is slow and scanning. He takes some time to commence a sentence. His sight is defective. He cannot stand. He has talipes equino-varus, but little of the latter. The equinus is very well marked and cannot be reduced with the legs extended. Both knee-jerks are exaggerated. The plantar reflexes are extensor and actively respond. Both feet are very blue and very fat. The arms are natural; he can feed himself well. Tâche cérébrale is well marked. His organs are healthy. The teeth are in good order.

(*Exhibited November the* 18*th*, 1905.)

21. CASES OF PSEUDO-HYPERTROPHIC PARALYSIS.

By GEORGE CARPENTER, M.D.

Case 1.—Charles T—, aged 11 years, was sent to me by Dr. Harrison Davison of Odiham, Hants, and was admitted into the North-Eastern Hospital for Children on August the 23rd, 1905.

He is said to have been weak since birth, and has never walked. At 2½ years old he used to get attacks of "stiffening of the limbs." The arms have been wasting for two or three months, but no difference has been noticed in the legs. For two or three years his mother noticed that when the boy stooped he had great difficulty in getting up, and she thinks he has been growing generally weaker for two or three months. No history of any infectious fever, and no history of any similar troubles in the family. The mother has had eight children, six of whom are living; the others died in infancy, one of convulsions, the other of diarrhœa.

On admission.—He can walk feebly with assistance on his toes and with the legs wide apart, but he could not stand alone, he is too weak to get up from the floor. He can raise himself from the floor on his hands and knees, but no further. When once up he can sit up unaided. When standing lordosis is marked, and the scapulæ are winged.

The knee-jerks are absent. The deltoids are decidedly enlarged and weak and the triceps muscles slightly enlarged and weak. The supra- and infra-spinati are wasted, the latissimus dorsi cannot be felt, and the trapezius muscles and the pectorals and biceps and coraco-brachialis muscles are also wasted. The calf muscles are enlarged and the muscles of the fronts of the legs rather prominent. The hamstring muscles seem to be infiltrated, and the muscles of the fronts of the thighs suggest that the boy is well developed muscularly, whereas his performances prove him to be very weak.

There are no fibrillary contractions. There is some loss of reaction to faradism, but otherwise the electrical reactions are normal, as the muscles of the legs, the arms, the shoulders, and the back.

Portions of the calf and pectoral muscles were removed under an anæsthetic. They appeared to be light in colour, but did not seem to be composed of fat. Dr. Theodore Fisher stained the end plates with chloride of gold and found them to be normal, and the muscle-fibres showed no structural alterations when stained with Sudan iii or when subjected to other micro-chemical processes.

The boy is fat in the face and fatuous-looking. He is not bright but he is shy, and is not so foolish as he appears to be, and he has received no education. He can name correctly most farmyard objects in picture-books and so on. His teeth are very yellow and ridged, except the left lower canine, which is quite white. The feet are cold. The plantar response is flexor.

No changes in the fundus oculi. He has incontinence of urine and fæces at times, but he asks occasionally.

Case 2.—Albert S—, aged 6 years, was admitted into the North-Eastern Hospital for Children on November the

2nd, 1905, for weakness of the legs and back of a year's duration.

He had measles three years previously. No family history of the affliction.

On admission.—The infra-spinati muscles are enlarged on both sides, as also both triceps muscles. The pectoral muscles are thin. The deltoids are somewhat lumpish, the right being the most marked. The calf muscles are decidedly enlarged, and the extensor muscles of the thigh are somewhat enlarged, the right more than the left. The knee-jerks are present. Plantar responses are flexor. The walk is high-stepping to clear the toes from the ground, and he gets up from the floor in the typical position, viz. by "climbing up himself." He has incontinence of urine and fæces always. There are no fibrillary contractions. He is a bright and intelligent and well-nourished lad, and his appetite is good.

(*Exhibited November the* 18*th,* 1905.)

22. A CASE OF CIRRHOSIS OF THE LUNG AND BRONCHIECTASIS.

By GEORGE CARPENTER, M.D.

John B—, aged 3 years and 9 months, was admitted into the North-Eastern Hospital for Children for cough on October the 9th, 1905.

He had measles when three months old and has coughed since; scarlet fever and whooping-cough in April, 1905. At five months old he was an out-patient at the North-Eastern Hospital, when he used to bring up "blood." At this time his fingers were clubbed and they have been getting worse. No family history of phthisis or syphilis.

On admission into the hospital he was a cyanosed child with prominent eyes and enlarged veins in the neck, and troubled with a paroxysmal cough much like that of whooping cough. He weighed 21 lbs. 4 oz. The liver and spleen were enlarged two fingers' breadth below the costal margin. The left side of the chest was absolutely dull from apex to base, back and front, and a cyrtometer tracing of the chest showed shrinking of the left side with a rounded outline. The left side was dull all over back, front, and axilla.

Broncophony was heard all over the front of the chest, together with tubular breathing and some medium-sized consonating râles about the left nipple. In the axilla there was broncophony and tubular breathing. Below there was broncophony and tubular breathing over the infraspinatus area and above the angle of the scapula. Over the lower half were medium-sized consonating râles.

Microscopical examination of the expectoration showed mucus and pus cells, both mononuclear and multinuclear. The micro-organisms were chiefly diplococci, without capsules, and short chains of streptococci. To the X rays there was no displacement of the heart to the right, and the lung was opaque from apex to base.

Clubbing of the fingers and toes was a marked feature, and the phalanges and metacarpal bones of the hands appeared to be thickened. Cardiac impulse was in the fourth interspace in the nipple line, and there was extension of dulness to the right.

A blood count showed red corpuscles 8,270,000 per c.mm. and white corpuscles 11,250.

Since he has been in hospital his weight has increased and is now the same as on admission. In October he lost $\frac{1}{2}$ a pound and then gained 1 pound in weight, and he expectorated from 1 to 6 ounces daily—usually from 2 to 4 ounces. The temperature in the early part of the month was normal, with occasional evening rises from 100° F. to 103° F., but at the end of the month there were daily evening rises, once to 104·2° F. During this month (November) evening rises have been of common occurrence—once to 105° F. Expectoration has been much about the same. (*Exhibited November the* 20*th,* 1905.)

23. FRAGILITAS OSSIUM SYPHILITICA, WITH MULTIPLE FRACTURES, PROBABLY CONGENITAL.

By LEONARD GUTHRIE, M.D.

MALE infant, aged 2 months. At birth the feet were noticed to be turned in, in a peculiar manner, but no other abnormality was observed. A week after birth swellings appeared about the left arm and both legs, which were so tender on handling that the mother was afraid to bathe the infant.

On admission.—A puny infant with shock of thick dark hair, cranial bones thin and widely separated. Well-marked syphilitic ulceration of buttocks. Liver and spleen not enlarged. Severe purulent ophthalmia present.

Skiagrams showed fractures of both femora, both tibiæ, right humerus and radius, left humerus and ulna. Considerable amount of callus about middle of left humerus and left femur, with angular deformity. The fracture of the left tibia was ununited, crepitus being obtained.

Under mercurial treatment tenderness of limbs soon subsided, ulceration of buttocks healed, ophthalmia ceased, and all fractures seem to have become united, though considerable deformity remains.

Family history.—Father and mother healthy; father is a sailor. Two sisters healthy. A brother died, aged 5 years, apparently from hydrocephalus. No miscarriages.

There is no evidence to show whether the fractures were intra-uterine or sustained at birth, nor any history of subsequent traumatism to account for them. Most of the epiphyses show globular enlargement, but none is separated from its shaft. The case is probably one of syphilitic fragility of the long bones rather than one of congenital rickets.

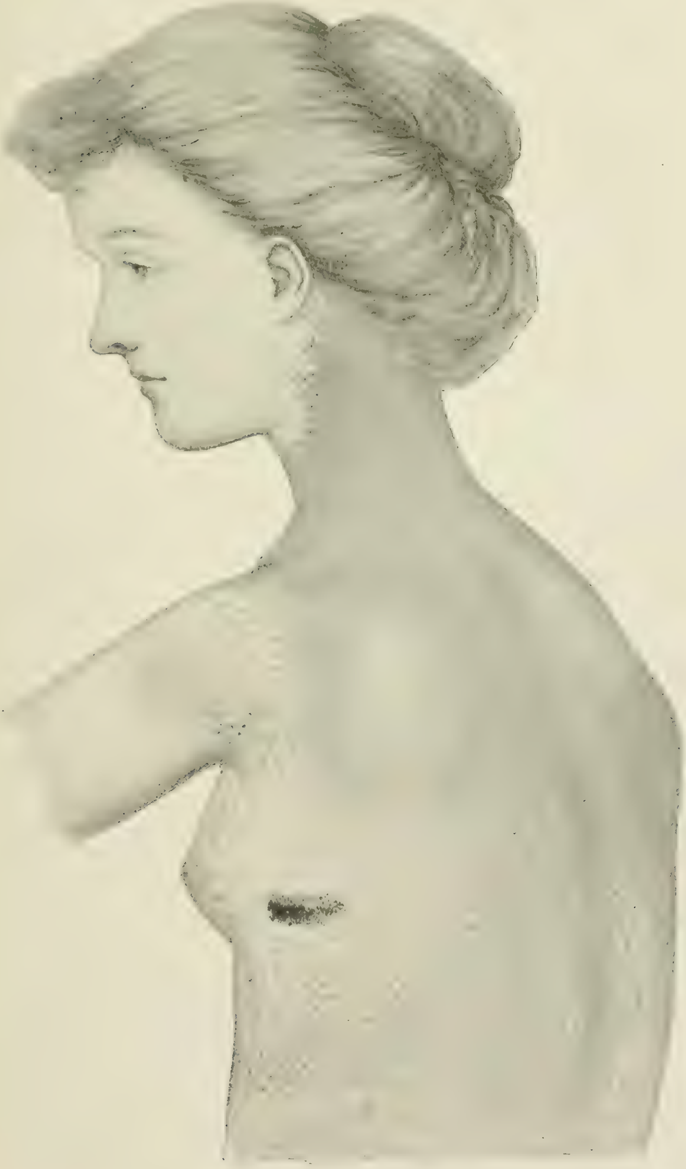
(*Exhibited November the 20th,* 1905.)

24. A CASE OF PECULIAR PIGMENTATION OF THE SKIN.

By F. PARKES WEBER, M.D.

The patient is a fairly well-developed girl, aged 15 years. The pigmentation affects the trunk and neck chiefly, and the extremities to a much lesser degree, the face being almost free. There are no areas of leucodermia. There is no evidence of urticaria, factitious urticaria, purpura, or any kind of erythema in connection with the condition. The patient presents, roughly speaking, three different kinds of cutaneous pigmentation, namely: (1) Diffuse brownish patches, especially a large patch over the upper part of the back and neck, which has a sharply defined, probably stationary, upper border, but is ill-defined below, in which direction it is apparently spreading and merges gradually into the ordinary skin. (2) Brown spots and small patches plentifully scattered over the trunk, the paler ones being probably those most recently developed. (3) A group of very dark, almost black, spots on the left side of the thorax, resembling a group of "pigment nævi," but not raised above the general level of the skin. These blackish spots were first noticed about three years ago. The earliest pigmentation noticed by the mother was at the back of the neck when the child was only about eighteen months old. Since that time the rest of the pigmentation has gradually developed, and fresh, relatively faint spots and patches have lately appeared on the extremities. The patient's general nutrition is fairly good, and menstruation has recently commenced. Though she looks rather pale, examination of the blood (Dr. Schenck) has shown nothing of pathological significance. The brachial blood-pressure by the Riva-Rocci instrument was found to be about 130 mm. mercury. The examination of the thoracic and abdominal viscera and of the urine shows nothing abnormal. Cutane-

Fig. 4.



ous sensation, knee-jerks, and plantar reflexes are natural. The mental development and general intelligence are up to the average. There is no pigmentation in the mucous membrane of the mouth. In the past history of the patient the only noteworthy point is that she has always been very liable to headache and bilious vomiting. But there has been no tendency to fainting such as to suggest Addison's disease. On the contrary, the attacks of vomiting seem to have been induced by certain articles of food, such as rich, fatty things and, at one time, raw apples. Moreover, the patient's mother, as a child and young woman, used to be subject to so-called "bilious attacks" (it may be noted in this connection that the mother has small patches of Xanthelasma palpebrarum, which appeared some years ago), and one or two others in the family have been inclined to similar attacks. Apparently no anomalous pigmentation has been observed in the family except in the patient herself. In her case, from the diagnostic point of view, it is to be noted that the pigmentation affects chiefly the covered parts of the body, and that there is no special liability to freckling from exposure to the sun. This is the reverse of what occurs in cases of Kaposi's Xerodermia pigmentosa. The variety in the colour and form of the areas of pigmentation and their situation (mainly on the trunk) make it seem most probable that the present case is allied to (that is to say, a *forme fruste*, or incomplete form of) Recklinghausen's disease—in fact, that it is a case of neuro-fibromatosis, with typical cutaneous pigmentation, but, as yet, practically without any (superficial) tumours. There is only a single small, flaccid, molluscous tumour to be found. This is situated on the lower part of the back and was first noticed about three years ago. Probably no other satisfactory explanation for the pigmentation can be offered except that mentioned above, which was first suggested by Dr. A. Whitfield when he was told about the case.

(*Exhibited November the* 17*th*, 1905.)

25. SOME POINTS IN THE DIET OF CHILDREN DURING THE SECOND YEAR.

By J. S. MACKINTOSH.

Much attention has been given as to the most suitable methods of feeding a child during its first year, but the feeding during the second year has not, perhaps, yet received the attention it deserves.

The family practitioner has now learnt the value of percentages, if he has not risen to the level of writing prescriptions for the Walker-Gordon milk laboratory, and when he has steered the child of a delicate mother to apparent robust health at the end of twelve months he believes he has vindicated the power of environment over heredity. During the next few months, however, the child, in spite of the most careful directions, often fails to thrive. It becomes thin and flabby, with troublesome constipation, and is prone to develop catarrhs and "liver" attacks. Worst trouble of all, it refuses to take an adequate quantity of milk, a very serious matter should acute disease attack the child. It is easy to ascribe these troubles to the reasserted power of heredity over environment, but an explanation of them may be found in faulty dieting, the causes being the administration of solutions of meat extractives (beef-tea, bovril, meat soups, etc.) and an undue preponderance of starchy food in the meals.

Nevertheless, the administration of solutions of meat extractives after weaning-time is sanctioned by most authorities on children's diets—*e.g.* Eustace Smith, Cautley, Hutchison, Starr, Crozier Griffith, George Carpenter, Marfan, Emmett Holt, Monti (of Vienna), and others—in most cases to be mixed with starchy food, such as bread or potatoes.

The following considerations may, however, be urged against their use: they have little or no nutritive value;

their strong flavour makes a child disdain the bland taste of milk, and gives it a vicious craving comparable to that of alcoholism; when mixed with farinaceous food the latter is imperfectly masticated, the palate being induced to treat each mouthful as proteid in nature; the gastric juice is uselessly stimulated; the child is induced to take farinaceous food beyond its natural inclination; it is apt to develop excitability and irritability, often wrongly ascribed to other causes; conditions of intestinal toxæmia, and possibly a tendency to convulsions are favoured.

A child can be maintained in robust health until eighteen months old on a purely milk diet with an occasional bone or crust to gnaw. Raw meat juice with its high percentage (5 per cent.) of proteid and its comparative tastelessness, and even scraped raw meat, are occasionally useful in weakly children or where special indications exist. Raw meat by its property of sticking to the teeth necessitates thorough mastication, and so development of the masticatory structures. Sloppy farinaceous foods are to be shunned as inducing lax habits of mastication and favouring gastric fermentation. All farinaceous foods should be strictly limited until the approach of the third year. Yolk of egg seems to suit some children at twelve months, but certainly not others. A little orange- or grape-juice occasionally is useful as an antiscorbutic.

(*Read November the* 18*th*, 1905.)

Discussion.

Mr. Milner Burgess (Harlesden) said he agreed with Mr. Mackintosh with regard to keeping to milk diet during the period mentioned. By that means there was less scurvy and rickets. Mr. Mackintosh did not touch upon what was even a more important matter, namely keeping the child warm. As a rule, he believed, children were not kept warm enough. The catarrhs of childhood were answerable, in his opinion, for more ills even than injudicious feeding.

Dr. Edmund Cautley said he could confirm practically all that Mr. Mackintosh had said as to the injury caused by the excessive use of meat extracts; but he thought that the mere fact that the child was given a little gravy, especially from uncooked meat, or occasionally beef-tea or soup, if it were given as prescribed, namely once in two or three days, would not be sufficient to account for all the symptoms which had been described. But he was sure it was an injurious habit, and one largely indulged in by mothers, to give strong beef-tea. It was not only given with potatoes and stale bread crumbs, but often alone. So, too, bovril and Valentine's extract impaired the taste for milk and carbohydrate foods. He (Dr. Cautley) did not think infants, as a rule, did very well on a milk diet during their second year. Japanese children seemed to do well on milk for two years, and they were generally breast-fed for that time. Still, the average child required some carbohydrate food, and that with milk must be relied upon until the fifteenth month. The white or yolk of an egg could be added. A more proteid diet, such as pounded fish or meat, could be given if the child were flagging and suffering from excess of carbohydrates. Many children were given much too large a quantity of sugar, which was frequently added to their other food, and carbohydrate dyspepsia was not at all uncommon in such children, the symptoms being a distended abdomen, flatulent dyspepsia, general malnutrition, and possibly wasting. He did not think the wasting in children of that age was always due to that cause. Most of the babies of the social class referred to by Mr. Mackintosh were overfed, and the mothers were proud of the fact that they were fat. Sometimes these children wasted because at that age they grew rapidly. On the whole, he thought Mr. Mackintosh was right in his contention as to the evils of nitrogenous extracts—for they were not really foods. Still, perhaps undue importance had been attached to a moderate use of such foods, for a little meat-juice given at dinner in the middle of the day did no harm.

Dr. F. J. Poynton supported Dr. Cautley's remarks and said one could not lay down any very general rule as to what particular food a child should have in the second year. Though he would agree that milk was most admirable for such children, there were many cases in which children did much better when allowed a little more than this ; the juice of red meat was good, patent meat extracts were not comparable in his opinion. It was also useful to give fat in the form of a little lightly boiled yolk of egg. He had seen much good come from that even before the second year.

26. A SACCULAR DILATATION OF THE SMALL INTESTINE.

By EDMUND CAUTLEY, M.D.

This remarkable specimen was removed from a female infant who was admitted into the Metropolitan Hospital under my care on April the 27th and died on May the 14th, 1905, aged 5 weeks.

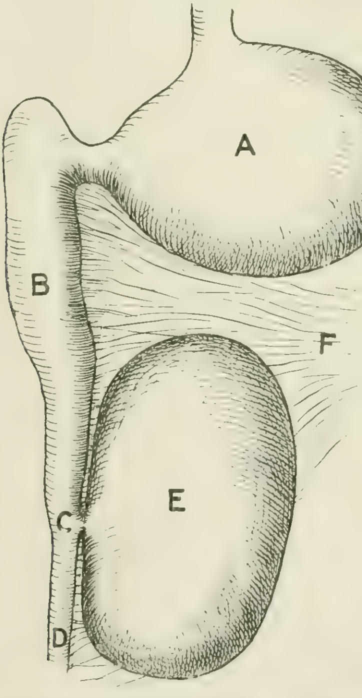
She was the first child, born at full-term, of healthy parents, but rather small. Vomiting began on the third day of life. It persisted, and was associated with diarrhœa after the seventh day. The vomiting followed every meal. Both the vomited matter and the stools were green. The child was bottle-fed on Nestlé's milk and Ridge's food.

On admission she was somewhat dusky, small, ill-nourished, and weighed 4 lbs. 13 oz. Nothing abnormal was found in heart or lungs. The abdomen was unduly prominent on the right side and a tumour, about the size of a pullet's egg, could be felt to the right of and on a level with the umbilicus. The tumour presented very peculiar features. It was fairly movable, elastic, sometimes dull and at others resonant on percussion, and it would disappear entirely after moderate gentle manipulation. There was complete absence of liver dulness in front. An inguinal hernia was present on the left side.

On May the 3rd the condition was unchanged except that there had been no diarrhœa since admission. The stools were small, formed, and green. Vomiting was quite infrequent. As the result of a diet of whey and small doses of cocaine the vomiting ceased for a few days, the stools became almost normal, and the child took food well. On May the 7th she weighed 5 lbs. 2 oz. On May the 12th the vomiting recommenced but stopped after two doses of cocaine and a return to whey. She had been taking weak milk and water for two days.

Apparently she was going on well, but on the 14th she had a convulsion, with cyanosis, carpopedal spasm, and unconsciousness. After about ten minutes she revived. Next day the breathing was very feeble and the child cold. There was no vomiting or diarrhœa. Death resulted from asthenia.

Fig. 5.



Diagrammatic representation of the sac. A. Stomach. B. Hypertrophied and dilated duodenum and jejunum. C. Constriction at level of opening of the sac. D. Thin gut below the constriction. E. Sac. F. Mesentery.

Post-mortem examination.—On opening the abdomen a cyst with opaque walls was seen immediately below the liver. The transverse colon was pushed downwards and the liver upwards so that the latter was not visible. The liver was very much flattened and the gall-bladder small, its tip being an inch and a half from the anterior edge of the liver. The cyst measured roughly three inches in the vertical, two in the transverse, and one in the antero-posterior diameters. It was situated in the mesentery, arising from the gut at a distance of fifteen inches from the pylorus. It communicated with the intestine by two adjacent openings close together, the larger one as big as a threepenny-piece and the smaller one quite minute. The edges of the openings were smooth and irregular. In general appearance the cyst can best be described as similar to a saccular aneurysm of the aorta with a small orifice.

The intestine above the origin of the cyst was dilated and very greatly hypertrophied, and below it was thin-walled and collapsed. Opposite the cyst the lumen of the canal appeared somewhat narrowed from pressure, though there was no true stricture. Both cyst and intestine contained normal bile-stained contents.

The wall of the cyst was, microscopically, identical in structure with that of the intestine from which it arose, the muscular coats being considerably hypertrophied.

Thus the post-mortem appearances clearly explained the symptoms present during life. The vomiting was due to pressure of the cyst, when full, on the lumen of the canal, causing intestinal obstruction. The alterations in resonance, the movable character, and the disappearance of the tumour on manipulation were clearly due to the variable amount of distension of the cyst and the gradual emptying of it by gentle pressure. This probably explains the variable degree of vomiting. When the cyst was more or less empty the obstruction produced by pressure of the cyst on the intestine would be absent.

I take it that there was no discredit in not making an accurate diagnosis during life, but I feel that possibly this child's life might have been saved by operation. Naturally, exact diagnosis was extremely difficult.

The history of persistent vomiting, beginning a few days after birth, suggested intestinal obstruction.

Congenital hypertrophic stenosis of the pylorus was excluded on account of the character of the vomiting and the presence of bile in the vomit. Moreover, such a diagnosis would not explain the presence of the tumour. After careful consideration I took the view that the case was one of incomplete stenosis of the gut, fairly high up, due to congenital defect of development, and that the variable tumour was due to dilatation of the gut above the stricture, increased at times by peristaltic contraction. Its disappearance I regarded as due to the manipulation forcing intestinal gas and other contents through the narrowed channel.

The fact that a diet of whey could be retained and produced a marked diminution of vomiting indicated that there was a reasonably patent channel. For this reason I postponed surgical interference in the hope that the child's condition would improve sufficiently for it to undergo operative treatment.

It is conceivable that a true mesenteric cyst may be produced in this manner, as a diverticulum of the intestine, eventually cut off from its connection with the gut. Gradually increasing distension would lead to thinning of the wall to such an extent that it might be impossible to recognise its structure. (*Exhibited November the* 18*th*, 1905.)

27. DIAPHRAGMATIC HERNIA.

By EDMUND CAUTLEY, M.D.

Diaphragmatic hernia is not remarkably rare, but sufficiently so to render each case interesting and worthy of record.

Paillard ('Thèse de Paris,' 1903) collected 481 cases, of which only 93 were on the right side. Hernia on the right side has been reported by Eustace Smith ('Lancet,' 1904, vol. i, p. 1424) in a boy of two years; by S. Gilford (*vide* 'Reports,' vol. v, 1905, p. 38) in a boy of four months and a girl of the same age; by A. Mori ('British Journal of

Children's Diseases,' vol. ii, 1905, p. 115) in a new-born infant. Mori also reports two cases on the left side in new-born infants, and Mougeot ('Bull. et Mém. de la Soc. Anat. de Paris,' November, 1904) a case in a girl who lived to the age of fifteen years.

Different varieties have been described in accordance with the period of development. In the "embryonic" variety there is no sac, for the hernia arises before the complete closure of the pleuro-peritoneal septum. It may be complete or incomplete, according as the whole or part of the dorso-lateral element of the diaphragm (pillar of Uskow) is absent. In the "congenital" or "fœtal" type the hernia has a sac. An "acquired" hernia is either a modified form of a congenital type, or a protrusion through an unusually large abnormal orifice.

The present case is one of the congenital variety on the right side.

Notes on the case.—The child, a female, was born on January the 17th, 1905, admitted into the Belgrave Hospital for Children on June the 2nd as a case of consolidation of the right lung, and died on June the 18th.

She was the ninth child, and born at full-time. Three children were dead. There was no family history of deformities or syphilis. Until six weeks before admission she was regarded as a healthy child, and had had no ailments. She was then brought to the hospital out-patient department after an attack of dyspnœa and cyanosis, and continued to attend for "bronchitis" and occasional attacks of dyspnœa. When seen in this department, on the day of admission, she presented marked recession of the chest, dyspnœa, cyanosis, and dulness over the right lower lobe of the lung. On examination in the ward two hours later she appeared in normal health. She was a mouth-breather, snored, and had a little cough. Examination of the chest showed recession, equal on both sides, a resonant percussion note all over, weak breath-sounds over the right base, and scattered rhonchi. The nasal obstruction was due to adenoids.

On June the 8th, as she continued in good health, the adenoids were removed under ethyl chloride anæsthesia. After this she breathed less noisily, but the recession of the lower ribs persisted. On June the 12th she vomited, and had distension of the abdomen. The percussion note over the right base was impaired. On the 14th there was no dulness, but the breath-sounds were so weak as to be almost inaudible, and she still had vomiting and abdominal distension. Two days later she had another attack of dyspnœa and was collapsed, after which partial dulness and weak breath-sounds were noted over the right base and in the axillæ. Recession was most on the right side. In the afternoon the dulness was more marked. The chest was explored with negative result, the needle apparently passing into an empty cavity in which it was freely movable. Vomiting continued next day, and on the following evening she became collapsed and died.

Necropsy.—The body was that of a well-nourished child of average size. On opening it the diaphragm was found depressed. The liver appeared rotated on its axis and the large lobe occupied the centre of the abdomen and reached down to the level of the umbilicus, the upper and anterior surface presenting. The stomach was much distended and filled the whole space between the liver and the symphysis pubis, except for a few coils of intestine in the right iliac region. The left lung was normal, except for some congestion; and the heart not displaced. The upper lobe of the right lung was compressed and pushed upwards, and the middle and lower lobes were small and undeveloped. The rest of the right pleural cavity was filled by a globular mass the size of a cricket-ball. This proved to be a subpleural hernia, containing almost the whole of the large intestine, the greater part of the small intestine, the left lobe of the liver, the gall-bladder, and part of the pancreas.

The most interesting points about this case were the variable percussion note, due perhaps to the varying contents of the gut and the position of the liver, and the absence of intestinal symptoms. There was vomiting during the last three days of life, but it was never excessive or suggestive of obstruction. The attacks of dyspnœa were not frequent, and the child seemed to have enjoyed good health.

(*Exhibited November the* 18*th*, 1905.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said both Dr. Cautley's cases would be most valuable additions to the records of the Society. The first must be extremely rare, and therefore probably no one would be prepared to discuss it. It seemed a pity that such excellent specimens should be relegated to the end of a busy meeting, and it raised the question whether it would not be well to have pathological evenings, at which only specimens might be shown.

28. A CASE OF MULTIPLE RENAL CALCULI IN A GIRL AGED SEVEN YEARS.

By FRANCIS JAFFREY.

The child has not had any previous serious illness. She is, however, a little deaf, and is said to have had otorrhœa. She has always lived in Lambeth.

For two years previous to the operation she has had severe attacks of pain in the left side, and these attacks became more frequent, and lately an attack every day, each attack lasting about five minutes. During an attack she was unable to pass urine, and was relieved as soon as she could pass it; there was no blood in the water. The pain came on by exercise, especially on skipping, and she was therefore disinclined to play with other children. She could not lie on her left side. On September the 8th she passed a small stone, smooth and facetted. Recently she became thin. There was no history of stone. An X-ray photo showed distinctly calculi in the left kidney.

On September the 18th Mr. Jaffrey operated by an oblique incision about 3 inches long, and on incising the lower part of the kidney removed eleven calculi rather larger than a pea. Recovery was uneventful.

(*Exhibited November the 18th,* 1905.)

29. A CASE OF REMOVAL OF A CALCULUS IMPACTED IN THE URETER CAUSING HYDRONEPHROSIS.

By R. CLEMENT LUCAS, B.S.

Mr. R. Clement Lucas related this case, which occurred in a boy, aged 11 years, who had had loin pain and attacks of colic for eight years—that is, since he was three years of age. Mr. Lucas opened the hydronephrosis at the pelvis of the kidney and failed to find a stone; but finding the orifice of the ureter dilated, extended his incision downwards and forwards to Poupart's ligament, and displacing the contents of the iliac fossa forward, found an elliptical stone imbedded in the ureter about two inches below the kidney. The stone was removed through a longitudinal incision which was closed with fine silk. The pelvis of the kidney was closed with chromic gut. No leakage followed, and the boy left the hospital healed and well on the sixteenth day after the operation.

(*Exhibited November the 18th,* 1905.)

30. PLEURAL EFFUSIONS, SEROUS AND PURULENT: PATHOLOGY AND BACTERIOLOGY.

By J. G. EMANUEL, M.D.

In thinking about pleural effusions in children the first point which confronts us is the great difference between the frequency of serous and purulent effusions in children and adults respectively. Gee (1) analysed 867 cases of pleurisy with effusion at all ages treated at St. Bartholomew's Hospital between the years 1884 and 1893 inclusive, and found that there were 169 cases occurring in children under ten years of age, and of these 84 were non-purulent and 85 were purulent, whereas of the 698 cases occurring in subjects over ten years of age, 567 were non-purulent and only 131 purulent—*i. e.* while in adults the serous effusions preponderated over the purulent in the ratio of 4 to 1, in children they were practically equal in number. Goodhart and Still (2) found that of 149 cases of pleural effusion occurring in children 71 were serous and 78 purulent.*

In cases occurring amongst children there is a good deal of disparity as to the influence of age on the serous or purulent nature of the effusion. Thus Holt (3) states that under the age of three years fluid is almost certainly purulent, and in this he is supported by Gee, who found that of the 78 cases under five years of age there were more than twice as many purulent as serous (53 to 25). On the other hand, of 58 cases examined by Goodhart and Still, the serous effusions preponderated over the purulent (32 to 26).

Empyemata in children are said to differ from those in adults in that the effusion in children is purulent from the first, whereas in adults it is more common for an empyema to originate from a serous effusion. With regard to this statement I will only point out that if we are going to compare an empyema of a child with one in an adult we must take pains to compare empyemata which are alike bacteriologically. We must compare two pneumococcal empyemata or two streptococcal empyemata—we must not draw comparisons between a pneumococcal and a streptococcal empyema, and in this connection we may remember that Netter found that while 53·6 per cent. of empyemata in children were pneumococcal, practically the same number in adults were due to the streptococcus. Netter (4) examined 109 cases at all ages and classified his results as follows :

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* Of 192 cases of pleural effusion in children under the care of George Carpenter, 85 were serous and 107 purulent ; see page 114.—Ed.

In adults:		
Streptococci	53·0	74·0
Streptococci and pneumococci	2·5	
Pneumococci	17·3	
Staphylococci	1·2	
Tubercle and putrid		26·0
In children:		
Pneumococci	53·6	74·8
Pneumococci and streptococci	3·6	
Streptococci	17·6	
Tubercle and putrid		25·2

In discussing the bacteriology of effusions it will be more convenient to speak of empyemata first, because in children purulent effusions are so much more common relatively than serous, and the bacteriological factor is easier to determine in purulent than in serous effusions; it is, in fact, for these reasons that most of the work on the bacteriology of effusions in children has been directed to the examination of empyemata. All observers are agreed that the organisms chiefly concerned in the empyemata of children are the pneumococcus, the streptococcus, the staphylococcus, and the tubercle bacillus. Of these, with rare exceptions, only the pneumococcus and the streptococcus are found pure.

Empyemata due to the pneumococcus.—About 75 per cent. of empyemata in children are due to the pneumococcus. This number is the average taken from 205 cases collected by the following observers: Netter (5) in Paris examined 66 cases and found the pneumococcus present in 65 per cent.; Koplik (6) in America examined 15 cases and found the pneumococcus present in 60 per cent.; Still at Great Ormond Street examined 15 cases and found the pneumococcus present in 93 per cent.; Blaker (7) at Shadwell examined 69 cases and found the pneumococcus present in 88 per cent.; and Bythell (8) at Manchester examined 40 cases, and found the pneumococcus present in 65 per cent. In the majority of cases the pneumococcus is found pure, but it may occur mixed with the streptococcus, the staphylococcus, the tubercle bacillus, Friedländer's bacillus, or the micrococcus tetragenus.

Cases of pneumococcal empyema may be divided into two classes: (*a*) empyema associated with pneumonia; and (*b*) empyema independent of pneumonia, the so-called primary empyemata.

(*a*) Lemoine (9) subdivided class (*a*) into: (1) Peri-pneumonic cases, cases accompanying pneumonia, in which there is a gradual effusion of purulent fluid in a case of pneumonia. (2) Meta-pneumonic cases, cases secondary to pneumonia, in which the pneumonia does not end by crisis but is followed by a short afebrile period, after which perhaps a sudden considerable rise of temperature will usher in an empyema, or, as the mother often expresses it, "the child was doing very well for two or three weeks after the pneumonia, when he seemed to fall back again." Lemoine says the secondary cases occur in debilitated subjects and the importance of distinguishing the two varieties lies in the fact that the peripneumonic variety, even though the effusion be large, is rapidly absorbed, while in the meta-pneumonic variety the duration of the disease is much more protracted. Blaker's experience, however, is directly opposed to this. He says that the meta-pneumonic cases do better than the peri-pneumonic, and this he attributes to the presence of incomplete resolution of consolidated lung in the peri-pneumonic, while in the meta-pneumonic cases the pneumonia has had time to undergo complete resolution before the occurrence of the empyema. A peri-pneumonic empyema may be said to be complicated by an unresolved pneumonia. In these pneumonic cases of empyema the pneumococcus probably reaches the pleural cavity by direct invasion from the consolidated lung. Bythell found abundant pneumococci in the lymphatic spaces in every layer of the subserous and serous portions of the pleura as well as throughout the lung.

(*b*) With regard to the so-called "primary empyemata" there are two views: (1) The empyema is really primary—*i. e.* is independent of any antecedent pneumonia. (2) The empyema is only apparently primary; it arises from an undiscovered patch of broncho-pneumonia and so differs in no essential respect from the pneumonic cases. Personally, I believe in the primary nature of these cases. Washbourn (10) in 1894 drew attention to such cases. He described three pneumococcal empyemata in which the symptoms resembled those of pneumonia—viz. a sudden onset, with rigor, labial herpes, high fever, cough, rapid breathing, and delirium. The following is a typical case which came under my own notice: An infant, aged 11 weeks, was admitted with signs and symptoms leading to a diagnosis of apical pneumonia of the right side of five days' duration. The infant died seven days after admission and at the necropsy an empyema containing from 4 to 5 ounces of pneumococcal pus was found and there were no signs of any pneumonic consolidation. Now, in such a case as this I cannot conceive of the existence at any time of even a small patch of consolidation. The entire illness only lasted twelve days, and during the whole of this time the patient was acutely ill and certainly in no condition to permit of the resolution of a patch of pneumonic consolidation, however small.

On theoretical grounds it is quite possible to conceive of the infection of the pleural cavity arising without the intervention of pneumonia, for we must remember that the pneumococcus is constantly present in the mouth and pharynx in health, and it may be that during a period of lowered resistance on the part of the individual it travels down the air passages, gains access to the pleura, and there sets up a suppurative inflammation without producing any appreciable change in the lung. To draw a comparison, von Behring has shown that the tubercle bacillus can pass through the intestinal mucous membrane without producing any visible change in the intestinal wall.

Empyemata due to the streptococcus.—Netter found that 17·6 per cent. of empyemata in children were caused by the streptococcus and 53 per cent. in adults. The streptococcus was found pure in only 4 per cent. of Blaker's and in 5 per cent. of Bythell's cases. While the streptococcus may occur alone, it is generally mixed with the pneumococcus, the staphylococcus, or the tubercle bacillus. Streptococcal empyemata may be (1) secondary to pneumonia, especially the pneumonia which complicates or follows the acute infectious fevers, measles, scarlet fever, and diphtheria. Councilman, Mallory, and Pearce (11) found from an examination of 220 fatal cases that the streptococcus alone or associated with other organisms was a common factor in the pleurisy of diphtheria and scarlet fever; (2), secondary to inflammatory lesions elsewhere than in the lungs—*e.g.* suppurative otitis media; or (3), may arise primarily like the primary pneumococcal empyemata.

Empyemata due to the staphylococcus.—The staphylococcus is not infrequently found in empyemata secondary to suppurations of other parts of the body—*e.g.* infective periostitis. It is a rare organism to find in primary empyemata, and this fact led Netter to state that if only staphylococci are found in a case of empyema it is probably *tuberculous* in origin and that more time is required to determine the presence of the tubercle bacillus than of the staphylococcus. Staphylococcus pyogenes aureus and albus were the commonest organisms found by Steele (12) in twenty-five cases and by Hervieux in fourteen cases of pleurisy in the newly born. These are cases occurring in the children of septic mothers and the infection is chiefly *viâ* the umbilical vein, though possibly the infection may arise from the milk. Such pleurisies occur, not only in the newly born, but arise at any period of embryonic life.

Empyemata due to the tubercle bacillus.—According to Netter 25 per cent. of empyemata in adults and children alike are due to the tubercle bacillus. Be this so, it is nevertheless very rare to find the tubercle bacillus in purulent effusions. According to some observers the absence of any micro-organism in an empyema points to its tuberculous origin, and on the other hand the presence of tubercle bacilli in an empyema is said to indicate a communication between the pleural cavity and a caseous focus in the lung or in a bronchial gland. Bythell suggests that different organisms have different powers of invasion of the pleural cavity from the lung, so that in a case of pulmonary tuberculosis complicated by an empyema it is far more likely to find a pneumococcal or streptococcal or staphylococcal empyema than a tuberculous one.

Many other organisms are occasionally found in empyemata. Several observers have found the bacillus typhosus both in serous and in purulent effusions occurring in the course of typhoid fever independently of any disease of the lung. Friedländer's bacillus, the bacillus coli communis, the diphtheria bacillus, and the micrococcus tetragenus have occasionally been observed. Putrid effusions are sometimes met with and in these organisms of putrefaction, especially proteus, are found. The exudates associated with malignant growths are generally sterile. In one of Bythell's cases, however, a pneumococcal empyema complicated a sarcoma of the lung.

The virulence of the organisms.—All observers are agreed that a pneumococcal is mild in comparison with a streptococcal or a tuberculous empyema. It must, however, be remembered that although the empyema is proved bacteriologically to be pneumococcal, it may in reality be complicating a pulmonary tuberculosis or even a sarcoma of the lung. Again, many streptococcal empyemata run a mild course. Considering pneumococcal apart from other empyemata, I would say that they exhibit three degrees of virulence:

(1) There is a mild variety, in which the pneumococcus remains localised to the pleural cavity. This is the case with the uncomplicated empyema.

(2) There is a more severe variety, in which the pneumococcus spreads from the pleural cavity, *viâ* the *lymphatics*, and gives rise by *direct extension* to pericarditis, peritonitis, or to empyema of the opposite side. This class includes those cases described by Frederick Taylor under the term "polyserositis" or "polyorromenitis," when they are due to the pneumococcus and when they begin in the pleural cavity.

(3) And finally, there is a very severe variety in which the pneumococcus gains access to the *blood-stream* and gives rise by *metastatic infection* to such complications as meningitis, cerebral abscess, endocarditis, subcutaneous abscess, and purulent arthritis. In this way arises pneumococcal septicæmia as a result of pneumococcal infection of a pleural cavity. In many cases of pneumococcal septicæmia an empyema occurs which is only the expression of a localisation of a general blood infection, but in some cases it appears as if the general septicæmia results from the empyema, and it is this class of case that I am including in the above description.

In connection with infection starting from the pleural cavity I would draw your attention to the not infrequent presence, in cases of pneumococcal polyserositis, of small collections of pus situated behind the parietal pleura, generally close to the vertebral column and about the level of the roots of the lungs. The presence of pus in such a position, I take it, is best explained by direct extension along lymphatics from the pleural cavity or possibly from infected bronchial glands; and although as a rule these collections are small in amount and consequently of little significance, at times they acquire such dimensions as to dominate the clinical features of the case, and I think then that the name "pseudo-empyema" might be applied to describe them. The following is a brief account of a case in point. A male child, aged 2 years and 2 months, was admitted into hospital on the day before his death, extremely emaciated, in a moribund condition, and with the history of having been ill with cough and shortness of breath for four months. At the necropsy, on opening the thorax it was seen that the mediastinum was displaced to the left. There was a considerable quantity of thin, purulent fluid in the pericardium and both layers of the pericardium were covered with a thick, villous, fibrinous deposit. Both pleural cavities were obliterated as the result of an acute fibrinous pleurisy, and the cause of the displacement of the heart at first was not obvious. It proved, however, to be due to a collection of 3½ ounces of creamy, greenish-yellow pus lying on the right side behind the parietal pleura, which had been stripped from the under-surface of the ribs along the entire posterior and lateral aspects of the thorax. The inflammation in the right pleura was acute and purulent, but the two layers were for the most part "glued together" by a recent fibrinous deposit. In the left pleural cavity the inflammation was acute but there was no pus. Further, on the left side, behind the parietal pleura, close to the spine, and just below the level of the root of the lung, was about half a drachm of pus similar in character to the very much larger collection lying behind the right pleura. The right lung was collapsed and airless; the left lung was crepitant. The pus from the pericardium, the right pleural cavity, and from the "pseudo-empyema" was pneumococcal.

Nuthall and Billington (14) have noted similar small post-parietal collections of pus in two cases of pneumococcal septicæmia.

Bythell has drawn attention to the amount of phagocytosis affording an indication of the prognosis. He found that in every case in which there was a good phagocytosis associated with a large number of well-stained organisms —irrespective of species—the symptoms were mild and the recovery rapid. On the other hand, phagocytosis was generally poor or absent in cases characterised by long and severe symptoms and also in those which ended fatally.

Bythell has also pointed out that the naked-eye appearance of the pus is not a trustworthy indication of the bacteria which it contains. In a typical pneumococcic empyema the pus is thick, viscid, of a pale greenish colour, and without any tendency to divide into layers. Now, while these characteristics were present in a majority of his pure pneumococcic cases, in a definite number the fluid was sero-purulent and rapidly separated into two layers. On the other hand, while streptococcic pus is typically thin, yellowish in colour, and on standing separates into two layers, the upper of which is clear and abundant, in one case due to the streptococcus in association with the staphylococcus and in another due to the streptococcus and the pneumococcus, the pus was thick, viscid, and exactly resembled pure pneumococcic pus.

The bacteriology of serous effusions might almost be dismissed with the statement that bacteriologically there is no essential difference between a serous and a purulent effusion. The same organism may produce at one time a serous and at another a purulent effusion. This is certainly true of the pneumococcus, the streptococcus, the staphylococcus, the tubercle bacillus, and the bacillus typhosus. Occasionally in cases of double pleurisy we find a serous effusion in one cavity and a purulent in the other, and cases of multiple effusions in the same pleural cavity are on record in which the contents of one loculus were purulent and that of another serous. Difficult as it sometimes is to determine the bacterial agent in an empyema, it is still more difficult to determine it in the case of serous effusions, for in the majority of these no micro-organism can be found by the staining of films made after centrifugalising a clear exudate, or by cultural methods, or even after inoculations have been made into animals. Again, in dealing with serous effusions, we are confronted with a difficulty that does not present itself in the case of empyemata, for given a clear effusion, it is not always easy to determine whether it be an inflammatory exudation (pleurisy with effusion) or a non-inflammatory transudation (hydrothorax). For example, a serous effusion makes its appearance in a case of Bright's disease or chronic valvular disease of the heart; we examine for micro-organisms, fail to find any, and assume it to be a passive transudation, whereas in reality it may be found to be a tuberculous pleurisy with a serous exudation but in which no micro-organisms could be found. On the other hand, in dealing with empyemata we may assume that all are inflammatory and associated with, if not actually due to, the action of micro-organisms.

The most important bacteriological work in connection with serous effusions has been carried out on so-called cases of idiopathic pleurisy, cases of pleurisy with sero-fibrinous exudation apparently due to chill occurring in subjects previously in good health and often of robust physique. Netter made inoculation experiments in fifty-five cases of serous effusion. Out of sixteen known to be tuberculous he obtained a positive result in eight; out of twenty-five supposed to be caused by exposure to cold, ten were proved to be tuberculous; and fourteen cases, due to rheumatism, pneumonia, cancer, or Bright's disease, gave negative results in every instance. Netter concludes as a result of this work that 40 per cent. of all primary exudates are tuberculous, and that in sero-fibrinous pleurisy the tubercle bacillus is the most common agent. Eichhorst, by using larger quantities of the exudate for inoculation—viz. 15 c.cm.—found evidence of tuberculosis in 62 per cent. Le Demany, by using still larger quantities (viz. from 10 to 50 c.cm., and sometimes as much as 300 c.cm.) demonstrated the tuberculous nature of forty-seven out of fifty-five cases of primary pleurisy—*i. e.* 85 per cent. Jousset (15) by allowing the fluid removed by tapping to stand, and digesting the coagulum with artificial gastric juice, centrifugalising, and then staining films, found tubercle bacilli present in every one of twenty cases of primary sero-fibrinous pleurisy. The results of many observations by different workers tend to the conclusion that of cases of idiopathic pleurisy perhaps so high a percentage as from seventy to seventy-five are tuberculous. On the other hand, Osler found out of 101 cases of acute pleurisy which he examined post mortem only thirty-two cases which were undoubtedly tuberculous.

A good deal of work has of late been done on the cytological examination of the exudate in these cases of idiopathic effusion, based on the observations of Widal and Ravant, and briefly the results may be stated to be: (1) in tuberculous effusions there is a preponderance of small mononuclear cells (lymphocytes); (2) in other inflammatory effusions the polymorphonuclear cells are the more abundant; and (3) in mechanical transudations the cells are fewer than in either of the foregoing varieties, and consist of flat endothelial cells, often in groups.

Probably a preponderance of lymphocytes points to a slight irritation, and as such irritation is generally tuberculous it may be accepted as a proof of the tuberculous nature of the pleurisy. On the other hand, a large number of polymorphonuclear cells points to a more acute irritation of the pleura, and therefore indicates a tuberculous pleurisy of recent origin, or if found late a pleurisy of a different nature. In this connection, however, we find contradictory statements. Thus, Gibson (15), of Edinburgh, says that "the relative proportion of the leucocytes is an expression of the stage and extent of the reaction, rather than a proof of the nature of its cause. During the early stages of all types the polymorphonuclear cells predominate, during the later stages the lymphocytes predominate." On the other hand, Hadley (16) stated at the British Medical Association meeting in 1905 that "the longer the effusion has been present the more polymorphonuclears there will be, so that even in tubercular cases of long standing the polymorphonuclear will be the more abundant cell. Indeed, a majority of lymphocytes go to prove tubercle, but a majority of polymorphonuclears does not prove the absence of tubercle if the case is of long standing."

With regard to serous effusion in children, in whom rheumatism in one or other of its varied manifestations is so common a disease, the question arises as to whether some of these primary effusions are not possibly rheumatic in origin. The practical difficulty in the solution of this problem lies in the fact that while rheumatic pleurisy is common it tends to be fibrinoplastic in character, and so rarely needs paracentesis. However, the first step has been taken, for Poynton and Paine (17) have found in pleural effusions the diplococcus associated with their names in cases of rheumatic polyserositis and have been able to produce rheumatic pleurisy experimentally.

Cytodiagnosis will not assist in distinguishing a tuberculous from a rheumatic effusion, for in both cases it is the rule to find a preponderance of lymphocytes. In such cases, however, I have been helped by an examination of the blood, for while in tuberculous pleurisies a leucopenia is found a moderate amount of leucocytosis is the rule in rheumatic pleurisies.

(*Read December the* 15*th*, 1905.)

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31. PLEURAL EFFUSIONS, SEROUS AND PURULENT: PATHOLOGY AND BACTERIOLOGY.

BY L. S. DUDGEON.

Mr. L. S. DUDGEON said that during the last five years he had made many examinations of the fluid taken from the chest in cases of serous and purulent effusion, and he thought that since Widal originally suggested this method it was one of the most valuable we have, because if one drew off clear effusion and found cells, either few or in large numbers, consisting entirely of lymphocytes, he thought one could definitely say it was tuberculous. If, on the other hand, one found what was described as the polynuclear phagocyte, one could say it was due to one of the pyogenic organisms. These remarks applied to cases which occurred in this country. Another point, to which reference was not made, was that in one case he found a very large number of cells which showed active mitotic figures, and when the child died it was found to have suffered from diffuse sarcoma of the chest cavity and pleura. With regard to the bacteriology of the affections under consideration, sufficient had already been said about the pneumococcus, and probably Dr. Blythell, who knew more about it, would speak on that presently. In those cases of empyema which went to the post-mortem room he in several instances drew off blood in a sterile glass syringe, and obtained an abundant growth of the pneumococcus, which suggested that the fatal cases of empyema might be examples of pneumococcic septicæmia.

With regard to another micro-organism, the staphylococcus pyogenes aureus, in four cases of bone abscess which developed empyema he obtained a pure culture of the staphylococcus pyogenes aureus. He thought it was time observers were more careful about speaking of streptococcic infections. It was extremely important, quite apart from bacteriology, because at the present day an antistreptococcic serum was given by some people very readily on the recognition of a few cocci forming a chain. It was well known that the antistreptococcic sera were made from virulent cultures of streptococcus pyogenes, and very often such a serum was given in cases which were not really streptococcus pyogenes infections. One of the most difficult matters in bacteriology at the present day was the identification of these "streptococci." He was at present investigating this subject with organisms obtained from scarlet fever and other "streptococci" obtained from cases occurring in a general hospital. It took from three weeks to a month to identify these streptococci, and though it was not of any particular value clinically, because the case would probably by that time be either dead or have recovered, yet in the future it would be of the utmost value. It was important that the profession should not label things as "streptococcic" too readily. With regard to the method which had been referred to of digesting the pleuritic effusions and then staining for tubercle bacilli, it was found that what were originally described as tubercle bacilli were nothing more than the exudate taking up the carbolfuchsin stain, and were not really bacilli. No reference was made to multiple empyemata. He remembered six cases when he was pathologist to the East London Hospital for Children, four of which went to the post-mortem room. All these cases had been operated upon and the chest drained, and doubt was expressed as to the reason they had died. In one case the empyema had been perfectly drained, but high up in the chest there was a clear effusion which had not been diagnosed. This case was of very great interest because there was an empyema filling up a large part of the chest, and there was also a perfectly clear effusion cut off by adhesions. Another example was a case in which between the concave surface of the base of the lung and the diaphragm there was a small collection of pus, absolutely shut off. In a third class—of which he had seen three examples—there was a collection of pus forming a second empyema, pointing down between the posterior border of the lung and lying on the vertebral column. They were all of extreme interest, because various causes, such as broncho-pneumonia, were thought to be the cause of the fatal result. The condition was really a localised empyema. There was another case in which an empyema followed appendicitis in a child, and it was the only example of it he had seen from post-mortem examination. From the pus which was drawn off during life he had obtained a pure culture of a virulent strain of bacillus coli.

(*Communicated December the* 15*th*, 1905.)

32. PLEURAL EFFUSIONS, SEROUS AND PURULENT: THE BACTERIOLOGY OF EMPYEMA FROM A CLINICAL POINT OF VIEW.

By W. J. S. BYTHELL, M.D.

About three years ago I was led to undertake the investigation which is briefly summarised here by the fact that only a comparatively small amount of attention had been devoted in England to the bacteriology of empyema in children. My results are, therefore, of interest chiefly in comparison with those obtained in other countries.

The objects at which I aimed were to determine (1) the source of infection of the pleura, (2) the species of micro-organism present, and (3) the influence of the bacteriology upon the clinical features of the disease. I therefore made a careful study of forty cases of empyema in children, the ages of the patients varying from ten months to eleven years. Of these forty cases, twenty-seven were males and thirteen were females.

As regards the *source of infection*, in over 80 per cent. of my cases this was undoubtedly an acute broncho-pneumonia, and in 10 per cent. there was an acute pneumococcic infection of a pre-existing pulmonary tuberculosis. In one other case the empyema followed upon a sarcoma of the lung, the patient eventually dying from acute pneumococcic meningitis. Thus, in over 90 per cent. the lung constituted the source of infection.

In only six cases did the empyema appear to be "primary"—that is to say, it was impossible to discover in these cases a definite source of infection outside the pleura. I agree with Netter that the majority of such cases are probably secondary to an overlooked pulmonary lesion.

The influence of age upon the *species of bacteria* is well known, and my results confirm very fully the great relative frequency of the pneumococcus which has been recorded by all previous observers as the distinguishing feature of empyemas in children. The actual percentage in my own cases—namely, 90 per cent.—is, in fact, somewhat higher than that found by Netter and others.

The results which I obtained were as follows:

	Cases.	Per cent.
Pneumococcus alone	26	65·0
Pneumococcus and streptococcus	1	2·5
Other (mixed) pneumococcic cases	9	22·5
Streptococcus alone	2	5·0
Streptococcus and staphylococcus	1	2·5
Friedländer's bacillus and staphylococcus	1	2·5
	40	100·0

The pneumococcus and the streptococcus were thus the only organisms found in a state of purity. One of them, moreover, was present in every case, except one which was due to the staphylococcus pyogenes albus and Friedländer's bacillus.

The complete list of micro-organisms met with was as follows:

	Cases.	Per cent.
Pneumococcus, alone	26	65·0
Pneumococcus, mixed	10	25·0
Streptococcus, alone	2	5·0
Streptococcus, mixed	2	5·0
Friedländer's bacillus, mixed	2	5·0
Staphylococcus pyogenes albus, mixed	1	10·0
S. pyogenes aureus, mixed	2	
S. cereus albus, mixed	1	
Tubercle bacillus, mixed	2	5·0
Micrococcus tetragenus, mixed	4	10·0
An unidentified bacillus, mixed	1	2·5

On comparison with the observations of Netter and others it will be seen that not only are the pneumococcic cases relatively more frequent, but that the proportion of streptococcic cases is somewhat below what is usually found even in children. Other points of interest are the presence of Friedländer's bacillus in two cases, and of the micrococcus tetragenus in four, also the total absence of putrid cases.

The question here arises as to whether the comparatively great frequency of the pneumococcus in my cases bears any relation to the unusually high proportion of post-pneumonic cases in the series. I found a pneumonic history, as I have already stated, in nearly 90 per cent., whereas others have in most instances recorded only about 50 per cent. I had other reasons also, which need not be entered into here, to believe that the pneumococcus was endowed with a particularly high degree of virulence during the period covered by this investigation. It must be remembered, moreover, that most of the previously recorded results were collected in warmer climates.

In the four examples of double infection of the lung by the pneumococcus and the tubercle bacillus, it may be added that in two instances the pneumococcus alone invaded the pleura, while in the other two both these organisms took part in producing the empyema. It is an interesting point as to which is the primary agent in these cases of concomitant infection. According to the views held by Landouzy and Babes they are to be explained by the assumption that the tubercle bacilli are previously present in the bronchial and mesenteric glands, both of which were found to be tuberculous in all my four cases after death and that the latent bacilli are stirred to activity by the pneumococcic infection. These writers believe that similar double infections are not uncommon at the commencement of pulmonary tuberculosis. These cases at least emphasise the importance of keeping in mind the possibility of a co-existing tubercular lesion of the lung when considering the prognosis of an empyema in a child.

Turning to the *clinical aspect of the bacteriology*, most writers are of the opinion that the pneumococcus produces a comparatively mild type of empyema, and that streptococcus is more apt to give rise to symptoms of much greater severity. My own observations tend to only partially confirm this view. In the first place, all my four streptococcic cases were very mild, and secondly, though the majority of the pneumococcic were milder than those in which other micro-organisms were present, yet several were distinguished by severe or long-continued symptoms. Moreover, six of these were complicated by a secondary invasion of other serous membranes, such as the pericardium or meninges, with a fatal result in three. The pneumococcus must therefore be regarded as possessing a very dangerous power of extending its operations beyond its original seat of infection. The frequency with which the pleura is invaded in pneumonia is in itself an example of this property.

The length of the interval between the onset of pneumonia and the formation of the empyema appears to have some relation to both the bacteriological characters of the pus and the clinical course of the disease. In the slowly-developed cases the micro-organisms usually gave indications of low vitality, while the symptoms were both milder and of shorter duration than in the cases where the pneumonia was rapidly followed by the infection of the pleura.

There are certain *other bacteriological points* to which I wish to call attention, as I am convinced that they are of considerable value in connection with the prognosis of the disease.

(1) If the pus contains but a small number of poorly-stained micro-organisms and gives feeble cultures, the prognosis is on the whole good.

(2) Abundant and well-stained micro-organisms, giving vigorous cultures, do not necessarily indicate a bad prognosis, especially when there is a large amount of phagocytosis to be seen under the microscope. If, however, the phagocytosis is poor or absent in such cases, the prognosis is nearly always bad.

(3) Vigorous cultures are not in themselves a sign of pathogenic activity.

On the whole, I look upon phagocytosis as the most reliable of these signs, as I have rarely seen any severe symptoms when the pus afforded evidence of active phagocytic action, while the latter was generally deficient or absent in the cases which were characterised by the length or severity of the symptoms, or which ended fatally.

In conclusion, I wish to add a word concerning the bacteriology in regard to *treatment.* In view of the mildness of the average pneumococcic empyema, many writers have advocated the removal of the fluid by simple aspiration. Apart, however, from the difficulty of carrying out a bacteriological examination of the pus in every case with which one has to deal, I am convinced that even in the mildest forms of empyema the large fibrinous masses that are left inside the pleural cavity by this method of treatment constitute a rich source of further infection and greatly increase the risk of grave or fatal complications outside the pleura.

I therefore consider that the plan of resecting a portion of a rib and inserting a drainage-tube should be adopted in every case, with perhaps the exception of those in which there are tuberculous lesions of the lung or pleura.

(*Read December the* 15*th,* 1905.)

33. PLEURAL EFFUSIONS, SEROUS AND PURULENT: SYMPTOMATOLOGY.

By G. A. SUTHERLAND, M.D.

If one has been following up the development of pleural effusion in a patient who has been suffering from acute pleurisy there is not as a rule much difficulty in making the diagnosis. But the problem before us to-day is, as I regard it, rather to consider the signs and symptoms in a patient who is seen for the first time and who is suffering from pleural effusion.

If there is a history of acute pulmonary trouble recently one may be directed to an examination of the chest. Such a history will frequently take the form of "an attack of congestion of the lungs," or "an attack of influenza," from which the patient had never thoroughly recovered. It may be some weeks or it may be some months after an attack of pneumonia before medical advice is sought, but an indefinite illness after pneumonia is always strongly suggestive of empyema. Many cases also occur in which there is no history of any previous illness. The only symptoms complained of or observed by the patient may be breathlessness and tiredness on exertion. When the patient is too young to describe them even these symptoms may have been overlooked. Coughing may be present, of a sharp, irritating, useless character, which often suggests to the listener the presence of pleural effusion. On the other hand, coughing may be entirely absent. The presence of anæmia is to be noted in all cases of pleural effusion of some weeks' duration. This is visible both in the skin and the mucous membranes.

In purulent effusion the anæmia shows itself as a peculiar yellowish-white or straw-coloured tinge of the skin of the face and the body generally, which is very suggestive of the underlying affection. Clubbing of the fingers, which has developed rapidly and without cyanosis, is another condition sometimes considered diagnostic of a purulent effusion. Although such a statement is too sweeping, the fact remains that the peculiar yellowish complexion and clubbing of the fingers and toes are more characteristic of empyema than of any other affection. Breathlessness is a symptom which varies in degree with the amount of effusion present. In an ordinary effusion the breathlessness may be present only on exertion. In a rapidly advancing effusion the breathlessness is much more marked than in a slowly progressive one. If very extensive effusion is present the breathlessness may be urgent and indicative of great danger to life. Wasting is not characteristic of simple serous effusion, but is frequently present in empyema. Dr. Goodhart lays stress on a puffy appearance of the face as indicative of a large effusion, simple or purulent. Venous engorgement of the face and other parts points to a large effusion with cardiac embarrassment. In an ordinary case of pleural effusion in a child there is no venous congestion. The temperature in both serous and purulent effusions may be normal, or slightly raised at night, or irregularly hectic; in short, there is no definite information to be gained from the temperature-chart.

While the general symptoms of pleural effusion are usually indefinite and uncertain, the physical signs in the chest are usually much more precise, and to a consideration of the latter I propose to devote the remainder of my time. Reference will be made only to cases of free fluid in the pleura, for the problems associated with localised pleurisy, or empyema, or bilateral effusions, or with a pleura previously affected with disease are quite different, and present much greater difficulty in the diagnosis.

There are certain physical changes in the lungs and surrounding tissue in the presence of pleural effusion, which were first described by Garland and Douglas Powell, and which are especially well manifested in childhood. We have to regard the chest as consisting of a bony framework, firm but flexible, and of two elastic bags, the lungs, which are everywhere unattached except at the roots. When the lungs are equally expanded the mediastinum, with the heart and other contents, is maintained in a central position, although not in any way immobile. The diaphragm is also a structure of great importance because it is the chief factor in the expansion of the lungs by its descent during inspiration.

How are these structures affected in the presence of, for example, a moderate effusion on the left side? The chest-wall on that side has been to a considerable extent thrown out of action, so that as an inspiratory factor it may be left out of account. The left lung has not been expanded as usual owing to the diminished movement on that side, and from its elastic resiliency has been shrinking before the increasing fluid, and naturally shrinking towards its one fixed point, the root. The diaphragm on that side has also suffered in its muscular force, so that on relaxation (expiration) it tends to follow the shrinking lung above it, and reach a higher level than normally. On the sound side of the chest the conditions are very different. There is a call here for fuller pulmonary expansion to counterbalance the effects of diminished respiratory power on the left side. The right side of the chest expands more, and the diaphragm contracts more powerfully on inspiration, with the result that the right lung is more fully distended, and its elastic tension is raised. This elastic tension, being unopposed by a similar tension in the left lung, will tell on the mediastinum and its contents, and tend to draw the heart over to the right side. If this tension in the lung is maintained for some time it will tend to draw towards the right side, not only the mediastinum, but also the left lung, and following the left lung, the diaphragm on that side will rise to a still higher level. The left side of the chest, unexpanded by active muscular movements, will be driven in by the atmospheric pressure.

Consider next what are the evidences of these changes as manifested on an examination of the chest. The deficient expansion of the left side of the chest will be shown but slightly, if at all, during normal (diaphragmatic) breathing. On forced breathing, however, it will be more evident. The chest-wall on that side is not expanded, but as time goes on it becomes flattened in the axillary region, with an outline more rectangular than cylindrical.

As regards the lung on the left side, the line of shrinkage will be approximately accurately shown by an arc of a circle drawn with the root of the lung as the centre, and the tip of the apex of the lung as the periphery. The root of the lung is located anteriorly at the junction of the second costal cartilage with the sternum, and posteriorly one inch to the left of the tip of the fourth spinous process. Expressed in other words, in the presence of pleural effusion the *lower* border of the lung is highest in the axillary region, and from this point slopes downwards and inwards anteriorly towards the sternum and downwards and inwards posteriorly towards the spine. The *upper* border of the lung is unaltered. On percussion over this lung area, the characteristic note is one of impaired resonance, which varies according to the amount of collapse present, and which may be described in different cases as slight, or boxy, or tympanitic. The note over this area is never flat. On auscultation the breath-sounds are diminished, the expiration is prolonged, and a bronchial element is frequently present over the area extending downwards from the root of the lung along the spine posteriorly and along the sternum anteriorly. Bronchial breathing is also frequently present at the extreme apex, and the observation of Dr. Goodhart, that when bronchial breathing is present at the apex in a child the base of the lung should be examined for evidence of fluid, is well worth keeping in mind. The area immediately above the lower margin of the lung may show the presence of some crepitations (hypostatic congestion) or friction sounds, but as a rule there are no accompaniments in the retracted lung. Vocal resonance is slightly increased all over and is often markedly so along the spine and the side of the sternum.

The position of the heart is of great importance. The heart is displaced towards the sound side, not pushed over by the fluid pressure, but drawn over by the unopposed elastic tension of the sound lung. When the effusion is on the left side the cardiac dulness will be found extending to the right of the sternum, while the apex beat is also moving to the right. In young children cardiac pulsation can often be felt below the ensiform cartilage, long before there is any evidence of engorgement of the right side of the heart. Visible and palpable cardiac pulsation in abnormal situations will often render clear, without the use of percussion, the altered position of the heart. The signs of cardiac displacement in children are more valuable than in the case of adults because they are manifested early in the disease, and are more easily determined.

As regards the area of fluid effusion, the upper border will be found to follow exactly the lower border of the retracting lung. It will, therefore, be highest in the axillary region and from there will slope downwards to meet the cardiac dulness anteriorly and downwards towards the spine posteriorly. The lower border posteriorly will be found to be that of the pleural sac. The lower border anteriorly is subject to variations, owing to the presence of the heart and the elevation of the diaphragm. Even in the presence of a considerable effusion the elevation of the diaphragm as shown by the stomach resonance may be considerable. One must not, therefore, assume that the presence of stomach resonance as high as the fourth or third rib in the axilla excludes the presence of free fluid in the pleura; it is, in fact, strongly suggestive of its presence. The characteristic signs of fluid as determined by percussion and auscultation are similar to those met with in adult life. The flatness on percussion, with board-like resistance, is present even with a small amount of fluid, but care should be taken that too firm percussion is not employed, as the note may then have a tympanitic character from the underlying stomach or lung. The breath sounds at the base posteriorly are faint and may be quite inaudible, with a corresponding diminution or absence of vocal resonance. Vocal fremitus is of little value as a test in childhood. On the other hand, there is frequently found in childhood bronchial breathing, with increased vocal resonance over the effusion. This may be faint and distant, audible only on deep breathing, or it may be as loud, and apparently as superficial, as over pulmonary consolidation. These differences would appear to depend on the condition of the underlying lung. In some cases, as in pneumonia or tuberculosis, the lung has been more or less solid at the time of the effusion, and resolution is delayed, so that the tracheal sounds pass *viâ* the bronchi through the solid lung and the fluid without much alteration. In other cases the lung, which was healthy at the onset of the effusion, becomes congested as it retracts, and may become so solid as to conduct the tracheal sounds effectively to the fluid. This latter conduction is usually found only in the presence of an extensive effusion.

So far we have been considering the signs of pleural effusion on the left side. The signs when the right side is affected are similar, except that the line of displacement is from right to left instead of from left to right. The stomach resonance is replaced by the hepatic dulness.

The signs above mentioned refer to the first stage of pleural effusion, the stage in which the fluid is not directly pressing on any of the surrounding structures, but is occupying the space formed by the receding lung. The second stage is reached when the elasticity of the lung is exhausted and the increase of the fluid leads to direct pressure on the lung, the heart, the diaphragm, and the chest wall. As regards the first two of these—the lungs and heart—the result will be an increased displacement in the same direction. As regards the diaphragm, the result will be that that muscle is pressed downwards, and the level of the stomach resonance descends. The chest wall, instead of being driven inwards by atmospheric pressure, will tend to bulge. Fortunately, these extreme conditions are but rarely seen now, as the symptoms tend to become more marked with the increase of fluid, and relief is sought and obtained before this dangerous stage is reached.

I believe that a careful examination of the chest ought to enable us, in the majority of cases, to form a definite opinion as to the presence of free fluid, without having recourse to the use of an exploring needle. The exact delimitation of the various thoracic organs is much more easily carried out in a child than in an adult, owing to the thinness of the surrounding tissues. We must not limit our investigations to one part of the chest but take it as a whole, and study the conditions and positions of the lungs, the heart, the diaphragm, the liver, and the area of effusion. By means of a careful physical examination we can, as a rule, distinguish between pleural effusion and (1) acute pneumonia, (2) tuberculous consolidation, (3) chronic fibroid phthisis, (4) new growth, (5) subphrenic fluid, and (6) pericardial effusion. Dr. George Carpenter and others have pointed out the risks of needling the chest whenever dulness is found. The great value of the exploring needle is to determine whether an effusion is serous or purulent, and this question cannot be definitely settled in any other way.

(*Read December the* 15*th*, 1905.)

34. PLEURAL EFFUSION, SEROUS AND PURULENT: MEDICAL TREATMENT.

By EDMUND HOBHOUSE, M.D.

When the Secretary first did me the honour of asking me to read a paper on this subject I was pleased to find that I was to have the first word, as it might be argued by some that the treatment of pleurisy with effusion was purely surgical. To begin at the beginning, there can be no doubt, I think, that we can cut short an acute pleurisy and prevent effusion by active measures in the early stage, but when once effusion has fairly commenced I am very doubtful whether it can be cut short by any medical measures; at least, I have never been able to satisfy myself that it was so in cases under my care. Nor do I feel it to be proved that when the effusion has reached its acme and become stationary we can hasten absorption much by counter-irritation and suchlike measures, though I am in favour of using them.

We may begin by eliminating from the medical side of the subject purulent effusions, in which the function of the physician so far as immediate treatment goes is to make the diagnosis (only rendered certain by exploratory puncture) and hand over the case without delay to the surgeon.

To come now to the treatment of serous effusions by medical means, the first thing is to put the patient to bed and to keep him there until all signs of definite effusion have disappeared. If this be done there is practically very little risk from a pleural effusion *per se,* but fatal accidents have occurred from patients moving about actively with a large collection of fluid in their chests. Pain is rarely present when effusion has once occurred; if it be, the use of counter-irritants is indicated, such as iodine or mustard-leaves. The diet should be light, and must be largely regulated by the temperature, no special restrictions being needed apart from fever. (The question of liquids will be discussed later.) Symptoms must be dealt with as they arise; sometimes cough is troublesome and has to be relieved by the ordinary remedies; sometimes nothing will relieve it except the removal of the fluid by paracentesis; as a rule, however, cough, like pain, is only troublesome in the early stage, before effusion has occurred.

As we are dealing *ex hypothesi* with cases of effusion, the main point is whether we can do anything to promote absorption by medical means, and, failing this, at what point paracentesis should be performed. We may further ask ourselves whether our practice as regards children will differ in any way from our practice as regards adults.

As to the promotion of the absorption of fluid by other means than puncture, this is a point which it is exceedingly difficult to determine with accuracy. If the fluid be serous the tendency is for it to be ultimately absorbed in a large number of cases (according to Louis, statistics quoted by West, 224 out of 227 cases got completely well without paracentesis, only one dying), but the rate of absorption varies in every case, and it is impossible to say what factors determine it. Sometimes it will begin suddenly when the level of the effusion has been stationary for a long time and then a large effusion may clear up in a few days. It becomes, therefore, exceedingly difficult to say whether this process has or has not been accelerated by drugs, administered outwardly or inwardly. To arrive at any conclusion would require a long series of cases treated on an uniform plan and carefully watched, and I know of none such. Two or three different lines of treatment have been pursued by different authorities, and it is necessary to consider them. All are based on what, in the language of the older therapeutists, were called either "derivative" or "alterative" methods. In the first instance it was sought to reduce the quantity of fluid by increasing the fluid output in other directions and reducing the intake. With this object diuretics, diaphoretics, and purgatives were given freely and a thirst diet adopted, the amount of fluid permitted being much reduced. While admitting that it is highly desirable to have all the emunctories acting freely, most authorities are agreed that the results attained by a rigid adoption of this system are by no means proportionate to the discomfort entailed and to the weakening of the patient which such vigorous treatment involves. The so-called "alterative" treatment consists in the administration of mercury and potassium iodide in moderate doses. Though called by a different name, the *modus operandi* is at least to some extent the same as that of the last method. Though I have no amount of evidence to go upon, I think that this is the most satisfactory treatment to adopt in cases where drug treatment is necessary, with the proviso that it be not applied to phthisical cases in an active condition.

The use of the salicylates was entirely based on the rheumatic theory of the origin of pleuritic effusions, and it is now held that a true rheumatic effusion is a very rare event. The question of the use of counter-irritants is one of some importance. In the case of children blisters may be definitely excluded as undesirable. The two milder preparations of iodine may be used or mustard-leaf. The best of all is, I venture to think, Linimentum hydrargyri, as to which I agree with the late Dr. Fagge and other good observers in believing that I have several times seen marked results from the application of mercurial ointment, or liniment, as I prefer. I am satisfied that it often has a remarkable effect in tubercular peritonitis, and it may be that its action is similar in pleuritic effusions. At all events, I think it should always be given a trial in all cases where no urgent reason exists for immediate paracentesis.

If a reasonable trial of these measures is insufficient to reduce the effusion it will be necessary to remove the fluid by puncture, and perhaps the most important point of all is the question when paracentesis is to be performed. It is impossible to lay down any hard-and-fast rules, every case must be considered on its merits, but there are certain general principles which it is well to bear in mind. It is undesirable to remove an effusion too early, as it is very likely to reaccumulate, and it is therefore better to wait until the febrile stage is over and the fluid has ceased to increase, unless—(1) there be signs of positive pressure within the thorax, such as bulging of the intercostal spaces, or (2) definite dyspnœa, a weak, irregular, and frequent pulse or lividity. If these be present in any marked degree there is only danger in delay. It is useless to specify exactly the size of effusion which will require removal, as these symptoms may arise with effusions of very varying size, and we must be guided rather by the condition of the patient than the actual amount of fluid present.

It is undesirable to leave a stationary effusion too long, even though many clear up in time without interference. If the effusion be of any size and has remained unaltered for a week, and things seem generally at a standstill, it is better to remove it. The removal of even a few ounces of fluid will frequently bring about the rapid absorption of the rest, apparently by altering the pressure relations in the thorax. Again, even though the amount of fluid be very small, it may keep up unpleasant symptoms for a long time and need removal. I have noticed in these cases a pulse frequent out of all proportion to the amount of fever; in one instance this persisted for many weeks and only disappeared on the removal of 6 ounces of clear serum.

There is one exception as to the rule about removing effusions, and that is in cases of well-marked phthisis the presence of the fluid seems to have a beneficial effect on the healing processes of the lung, and in old cases of phthisis there is some risk of producing hæmoptysis by removing it. This point does not often come into consideration in the case of children, but it is one that must be borne in mind. It does not apply, of course, to those effusions which seem so often to be the earliest signs of tubercle.

It has already been said that when the presence of pus has once been ascertained, the sooner it is removed the better, but without puncture it is impossible to be certain. We have recently had a very timely warning about the dangers of the indiscriminate use of the needle from the Editor of 'The British Journal of Children's Diseases,' Dr. George Carpenter, with which I cordially agree. I only wish to call your attention to this fact, viz. that in the case of children under five two out of three pleural effusions are purulent, and between five and ten one out of every two. I have taken the figures at St. Thomas's Hospital for the last ten years, and find that they agree closely with others in this respect, and it is well to bear this in mind. It is a strong argument against deferring exploration too long in cases where we feel fairly certain of the presence of fluid, though not for tapping every chest where there is a little dulness.

The further treatment of empyema and the various questions arising with regard to it I leave to my surgical colleague. I will merely add that I have no experience of the use of serum in streptococcal cases, though I should certainly try it in a suitable instance.

Some mention must be made of the after-treatment of cases of pleurisy in which effusion has occurred, as it is of very great importance to the future of the patient. It must be regarded as a very serious matter, and in many cases, especially where there is a family history of phthisis, as a warning of the possible onset of some tuberculous disease. Therefore after a week or ten days of the alterative treatment outlined above it is desirable to begin a course of tonics with cod-liver oil, according to the general condition of the patient, and to keep it up for some time. Respiratory exercises suitable for promoting the re-expansion of the lung should also be prescribed, as otherwise the chest movements on that side may remain very limited when adhesions form, as they almost always do.

Change of climate is very desirable and in the case of well-to-do children a winter abroad is often very valuable.

(*Read December the* 15*th,* 1905.)

35. PLEURAL EFFUSIONS, SEROUS AND PURULENT: SURGICAL TREATMENT.

By LOCKHART MUMMERY, B.C.

I THINK I may say that the surgical treatment of pleural effusion is confined entirely to the treatment of empyema, as although the treatment of simple pleural effusion is usually surgical and was in old times performed by surgeons, it is now exclusively practised by physicians. When I had the honour of being asked to open this discussion on the surgical treatment of pleural effusion I thought that it would be better not to enter into an exhaustive discussion upon the treatment of empyema, but simply to suggest those points in the treatment which seemed to me to be of importance, and especially those where there is room for improvement upon present practice. In a discussion of this sort what we chiefly want is an expression of opinion by those taking part in the discussion as to the more debatable points in the treatment of this condition.

The after-histories of cases of empyema which have been surgically treated is one of the results which it is to be hoped this discussion will bring forth. I have followed up the cases treated at the North-Eastern Hospital during 1904 and tabulated the results, but the number is too small to be of much value in drawing conclusions.

There were fifteen cases, with six deaths. Two of the children who died were not operated upon. Of the four others one had a double empyema, one died from the results of sepsis some time after operation (this child had been ill for two months before coming to the hospital), and the two other children died from complications, one from acute nephritis and one from pericarditis. Nine patients recovered and the result was perfect in four; that is to say, there was nothing but the scar left. In one of these there is a gap in the rib, but in the other three no gap can be felt. In two cases the result was fair, there being a slight difference in the expansion on the two sides of the chest. The result was unsatisfactory in two cases. In both these cases the empyema had been present for some considerable time before being operated on—in one case four months and in the other sixteen days.

Empyema occurring in children may be divided into two distinct classes, which are of particular importance from the aspect of treatment—(1) those cases in which the infection is a purely pneumococcal one, (2) those cases in which a septic, tubercular, or mixed infection is present. In all cases of empyema in children a bacteriological examination of the pus should, if possible, be made, though one can frequently tell roughly with what form of infection one is dealing by the appearance of the pus.

In most cases of infantile empyema the infection is a purely pneumococcal one, and from a surgical point of view little is required beyond allowing the pus to escape, and so enabling the lung to re-expand. Drainage for any length of time is in such cases entirely unnecessary, and by interfering with the re-expansion of the lung may be harmful. In the cases of pneumococcal infection the tube may usually be removed on the second or third day, and drainage for any longer period is seldom required. The chest remains open for a day or so longer, and it is not often that any re-accumulation occurs. In many cases I believe that incision without drainage is all that is necessary; this treatment was advocated by Dr. Sutherland in 1894. In cases of small empyema where the infection is pneumococcal, the patient's general condition is good, and there is reason to suppose that the elasticity of the lung has not been seriously impaired (that is to say, in those cases where rapid re-expansion of the lung may be expected), all that is necessary is to make a free opening in the chest by removal of a portion of rib through which all the fluid can escape. If then the lung is found to expand well, no drainage-tube need be inserted. Immediate re-expansion of the lung will usually take place as the result of coughing or crying, and this should be encouraged and the fluid assisted to escape by turning the patient on to the affected side.

In the few cases in which I have treated empyemata in children by this method it has been entirely successful, the wound has very rapidly closed, and no re-accumulation of pus has occurred. I think there can be little doubt that some re-accumulation must be looked for in a certain percentage of cases so treated, but this is not likely to be serious, and will merely necessitate re-opening the wound. This is the treatment now commonly adopted for psoas abscess, and the circumstances are far more favourable for the cure of an empyema by incision without drainage than for psoas abscess where there is diseased bone present. If the child's general condition is good and the infection not a severe one, any infective material in the pleural cavity which does not escape will almost certainly be dealt with satisfactorily by the pleura once the bulk of it is removed. In cases of mixed infection, or where the pleura is much thickened, or the child's general condition bad, I should prefer drainage.

There is one point in connection with the treatment of empyema which is, I think, too frequently neglected and which has an important bearing on the result. Too often an empyema is looked upon as a septic abscess and as therefore not requiring any special care as regards aseptic technique in its treatment. This is, of course, entirely wrong. As a rule the infection is a simple pneumococcal one, and secondary infection with other pathogenic organisms will greatly add to the severity of the condition and seriously retard recovery. Moreover, owing to the pleura being already damaged, infection will occur more easily, and will probably be more serious if it does occur.

I believe that when operating on an empyema the skin should be cleaned and the operation carried out as carefully as if the abdomen were going to be opened, and the wound should be subsequently treated with the greatest care to prevent any possibility of further infection.

You will perhaps think that it is unnecessary of me to emphasise this point, but I am certain that sufficient care is often not taken to keep the wound free from infection, with the result that an infection which was at first a purely pneumococcal one becomes after a few days a mixed infection.

In looking through the notes of cases of empyema one far too frequently sees on referring to the charts that a temperature which before the operation was but slightly raised became about three days after operation of the septic type, and I cannot but believe that this indicates secondary infection of the pleura.

In those cases where there is a mixed infection, where the pleura is much thickened, or for some other reason re-expansion of the lung is likely to be much delayed, good and proper drainage is essential. The best tubes I believe to be those designed by Mr. Bilton Pollard, with a double flange and just long enough to pass through the chest wall and no more. The method advised by Mr. Watson Cheyne, of placing a piece of green protective over the tube, or opening the chest wall so as to form a valve and prevent the ingress of air while it allows the pus to escape, is much to be recommended.

With regard to the best place for opening the chest. I think it is very difficult to lay down any rules. In cases of pneumococcal empyema, when prolonged drainage is not a serious consideration, the old-fashioned opening in the mid-axillary line is the most convenient. In the mixed and septic cases the posterior opening just below the scapular angle is much to be preferred on account of the much better drainage secured.

The anæsthetic.—The anæsthetic is a point deserving of special attention in the operation for empyema, as there can be no doubt that there is danger in the administration of an anæsthetic in cases where a large empyema exists. I think light anæsthesia with C.E. mixture or very light chloroform anæsthesia are best, and this should when possible be given combined with oxygen inhalation.

When dealing with a large empyema in a young child it is important not to allow the pus to escape too rapidly, and in some cases it is safer to slowly draw off the greater part of the pus with an aspirating needle. Mr. Godlee * recommends that if the chest is very full of fluid and the breathing and heart's action are hampered, a considerable quantity of the fluid should be drawn off before the anæsthetic is administered. I know of at least one case in which the child died directly the chest was opened and a large accumulation of pus allowed to escape rapidly.

In cases of foul empyema, and in those where there is much pyogenic membrane, I believe in irrigating the pleural cavity after making a free opening into the chest. The practice of irrigating the pleural cavity has been much criticised, and some surgeons have altogether condemned it owing to the fatal results which have sometimes followed. There can be little doubt, however, that these fatal consequences were due to the method of irrigation and the fluid used rather than to the practice itself.

Mr. Tubby some years ago performed some experiments upon dogs to elucidate this point, which he referred to in his paper on "Sudden Death in Children" last year. He found that no serious results followed irrigation of the pleural cavity with normal saline solution, but that irrigation with even quite weak solutions of carbolic acid or iodine often caused the immediate death of the animal. No surgeon would think of irrigating the abdominal cavity with carbolic or iodine solution, especially in children, but such solutions have been recommended from time to time for irrigating the pleural cavity in cases of empyema. I believe irrigation to be perfectly safe if normal salt solution or sterilised water is used and care is taken not to produce positive pressure in the chest.

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* Fowler and Godlee, 'Diseases of the Lungs.'

The chief advantage of irrigation is to wash out the large pieces of pyogenic membrane which are often present, and which, if allowed to remain, may act as foreign bodies and so keep up the discharge. While I do not think that irrigation should be employed as a routine measure, I think it has a decided field of usefulness in certain cases.

The operation for empyema is a comparatively simple one, and a successful result depends mainly upon two factors—(1) letting out the pus at the earliest possible moment, (2) careful after-treatment. There can be no doubt that the worst cases are those in which the empyema has existed for some considerable time before being operated upon. These are the cases which most frequently prove fatal, and also in which, if recovery occurs, it is most difficult to obtain proper re-expansion of the lung and rapid healing of the wound. It is, however, entirely a question of early diagnosis, and as the surgeon is a purely secondary agent in the treatment of empyema, and has merely to treat cases when they are referred to him, it does not come within his province. There is no doubt, however, that the operation should be performed directly the presence of pus within the pleural cavity has been diagnosed, and that any delay very seriously affects the result.

Everything should be done after the operation to allow and encourage easy respiration. This is a factor in aiding recovery which is sometimes overlooked in the desire to keep the child warm and keep the dressings in position. It is very easy in infants and young children to embarrass the respiratory movements by tight bandages or heavy bedclothes. The bedclothes should always be supported on a cradle, and the greatest care should be taken to see that the bandages do not in any way restrict the thoracic movements.

In the after-treatment of empyema everything depends upon obtaining rapid re-expansion of the lung after draining away the pus. The after-treatment should, I think, be divided into two periods.

The first is that during which an opening in the chest exists. Here we have to deal with a condition of pneumothorax, and, consequently, since expansion of the chest will no longer give rise to negative pressure in the pleural cavity on that side, we cannot avail ourselves of the ordinary breathing exercises, and we must make use of the positive pressure produced by the sound lung during expiration to expand the affected one. The patient should be made to expire forcibly against resistance. The simplest way of doing this is to make the child blow forcibly through a small tube held in the mouth, such as a piece of glass tubing. A good plan in a child is to give it a whistle or trumpet which will not make a noise unless blown hard.

During the second period we are no longer dealing with a pneumo-thorax, and our object is to obtain expansion of the lung by negative pressure in the pleural cavity as in normal respiration. This can only be done when the pleural cavity is closed by the healing of the wound, or by the formation of adhesions between the visceral and parietal layers of the pleura. At this stage in the after-treatment also we wish to develop and increase the musculature on the affected side of the chest in order to prevent any tendency to deformity. These objects can best be attained by suitable respiratory exercises, which, when possible, should be continued for some months after all symptoms have disappeared.

The best plan, when the parents can afford it, is for the child to attend regular classes at a surgical gymnasium, preferably on the Swedish principle, and conducted by a properly trained instructor. A skilled instructor is essential, and the child is much more likely to do the exercises properly and take an interest in them when in a class with other children than if alone. Special attention

Name.	Age.	Operation performed or not.	Duration of illness previous to operation.	Time when tube was removed.	Wound healed.	Result.
G. B—	4	+	8 weeks	10th day	6 weeks	Perfect.
R. B—	$1\frac{7}{12}$	—	—	—	—	Died.
E. K—	4	+	4 months	—	2 months	Bad. Mixed infection
W. H—	4	+	—	11th day	—	Perfect. Mixed infection.
D. M—	$2\frac{1}{2}$	+	2 months	7th day	-	Died (sepsis).
R. L—	2	+	4 days	—	—	Died (double empyema).
T. C—	2	—	3 weeks	--	—	Died.
E. R—	$1\frac{1}{2}$	+	2 days	—	—	Died (acute nephritis).
E. N—	2	+	2 days	—	—	Died (pericarditis).
M. D—	6	+	16 days	16th day	—	Poor. Some falling in of chest and bad expansion.
G. H—	9	+	2 weeks	17th day	—	Not known.
E. P—	$1\frac{1}{4}$	+	Pneumococcal infection at first, but became septic 3 days after operation			Very good result. Gap in ribs.
W. A—	$1\frac{1}{2}$	+	Mixed infection	—	—	Fair. Slight flattening of chest.
R. B—	6	+	1 week	Mixed infection	--	Perfect.
T. M—	$5\frac{1}{2}$	+	3 days	–	—	Fair. Slight flattening of chest.

should be paid to exercises which develop the chest and arm muscles, and those which increase the respiratory index. In very young children such exercises are, of course, impossible, but passive exercises, if properly done, may be of service in some cases.

It would be an excellent thing if in all children's hospitals classes for surgical exercises could be organised. Such classes, conducted by a skilled instructor, would cost but little, and would be of great benefit to children recovering from empyema and suffering from lateral curvature of the spine. I believe the time will come when a surgical gymnasium will be considered a necessary part of every children's hospital. Most Swedish hospitals and several German ones now have such gymnasia.

With regard to secondary operations in cases where the lung has failed to re-expand, I think the results of such operations as Estlander's are often far from pleasing; apart from the deformity of the chest which they produce, secondary spinal curvature and other troubles not infrequently result. It seems to me that much may be done by careful attention to the after-treatment to do away with the necessity for such operations. In any case, Estlander's operation should not be done too hastily, but plenty of time should be allowed to see if the lung will re-expand, and, if possible, suitable respiratory exercises should be carried out with the same object.

(*Read December the* 15*th*, 1905.)

36. PLEURAL EFFUSIONS, SEROUS AND PURULENT: PATHOLOGICAL AND CLINICAL OBSERVATIONS ON.

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For the purpose of this discussion and as a contribution to the study of pleurisy in childhood, I have searched my note-books and I have reviewed the clinical histories of 192 cases which have passed through my hands during the last few years. Of these 192 cases there were 115 males and 77 females.

The serous variety totalled 85—48 on the right side, 36 on the left, and 1 double.

The purulent effusions numbered 107—51 on the right side, 54 on the left, and 2 double. No less than 116 cases were 5 years of age or under, 77 of them being empyemas.

Of those with serous effusions 7 died, and of those with purulent effusions 32 died, 2 of them with lardaceous disease.

Of the 7 former 1 suffered from mediastinal new growth, 1 from pyæmia (bone), 1 from pneumonia, 3 from tuberculosis, and 1 died from exhaustion after several aspirations.

Of the 31 deaths from empyema there were 5 cases of pneumonia on the affected or on the healthy side, 2 had peritonitis in addition, and 1 tuberculous meningitis; 4

died from tuberculosis in one form or another; 1 from scarlatinal nephritis and exhaustion; 1 from interstitial nephritis; 2 from exhaustion; 1 with cheesy tracheal glands; 2 died suddenly; 2 died out of hospital; 2 had lardaceous disease; 1 was undiagnosed (coma and convulsions); 1 had pus in the abdomen; 1 pyæmia; in 4 post mortem was declined; 1 died of (?) peritonitis; 1 had fœtid pus; 1 succumbed on the operating table; 1 from washing out the cavity (convulsions and high temperature); 1 from abscess and gangrene of the lung; and 1 from multiple abscesses of the lung.

In a female, aged 2 years, with double empyema, and during life a swollen left hand and forearm and œdematous chest-wall, there was found a thrombus of the left innominate vein extending to the subclavian and internal jugular veins; she also had general tuberculosis.

In a boy, aged 3 years, with empyema, there was free air in the pleural cavity. The right lung was collapsed, the pleural sac being divided into two by dense adhesions provided with openings. A small abscess cavity in the lower lobe of the lung filled with caseous material communicated with a bronchus, but not with the pleura.

In a boy, aged 14 months, with empyema, the left lung was collapsed. In the left lower lobe of the lung, in which there was a small localised abscess, was an orifice connected with a superficial cavity and indirectly with the bronchi. On the under-surface of the lung were three small apertures communicating with a network of ragged cavities partially separated by bridges of shreddy tissue, and every here and there were portions of solid lung-tissue becoming gangrenous.

A male, aged 3¾ years, had a chocolate-coloured effusion which had not been detected, on the side opposite to the empyema; both lower lobes of the lungs were collapsed, and tuberculous, together with a condition of general tuberculosis.

A child, aged 6 years, cured of an empyema, subsequently died of tuberculous meningitis. The adhesions in the pleura were tuberculous, but there were no tubercles elsewhere.

Many of the cases commenced with a sudden sharp febrile attack ; some few gave a history of a preceding exposure to wet and cold—viz. 10 in 101 cases. Occasionally injury in the shape of a fall or other traumatism was apparently the starting-point.

Many cases originated in pneumonia, broncho-pneumonia, bronchitis, and pertussis followed by pneumonia. In many children a rheumatic or a tuberculous family history was obtained. Scarlatina, measles, rheumatism, tuberculosis of the lungs, typhoid fever, diphtheria, mumps, and varicella were the starting-points in others.

Symptomatology : initial stage.—For some not obvious reason, or following an exposure to wet and cold and usually in sequence to one of the conditions previously enumerated, the child is suddenly seized with the usual febrile symptoms accompanied by a temperature of 101° F., 102° F., 103° F., or 104° F., or even more.

There may be headache, vomiting, lassitude, shivering, drowsiness, or even convulsions. Fever is of variable duration—a day or two, perhaps a week, or even a month. Rarely there is delirium or, rarer still, a typhoid state.

Pain may be absent or very slight; occasionally it is acute and the affected side is tender. But pain may be complained of elsewhere, quite away from the seat of mischief. Thus it may be felt at the corresponding shoulder, be referred to the sternal, epigastric, umbilical, or hypogastric regions, be felt on the *opposite* side, or even in the limbs. In one child under my care with left-sided pleurisy it was experienced down the corresponding arm.

Sometimes there are profuse sweating and diarrhœa, but these symptoms belong rather to purulent pleurisy, though they are not confined to this variety.

The respirations are increased in frequency and are accompanied by laryngeal grunting and sometimes by dilatation of the alæ nasi. The pulse respiration ratio is also altered. When effusion is abundant the pulse becomes small, frequent, and irregular.

Cough is a feature and is frequent, short, hacking, and dry. Orthopnœa may occur during coughing, and cyanosis become marked at that time. In such a case the effusion of fluid may be very rapid and will be found to be copious. When effusion is abundant the pulse is small, frequent, and irregular. In young infants collapse is a noticeable feature, and in their case wasting is rapid.

Not infrequently the initial stages of the illness are not very pronounced, and the child is brought to the doctor for advice because it is wasting or because it has a poor appetite; on the other hand, its condition may be so weakened by the complaint that it has to be carried into the consulting-room, or it drags its feeble limbs there with difficulty. By physical examination alone can the nature of such a case be determined. In many instances pleurisy is not suspected, and is only discovered on making the usual routine physical examination. Pleurisy is often overshadowed by the general disease. Thus in pneumonia, broncho-pneumonia, and bronchitis the detection of a friction sound by the stethoscope may be the sole evidence of this complication. In some cases the physical signs of pneumonia are rapidly followed by those of pleural effusion, and then often of a purulent type. In many instances pleurisy though present escapes detection by the methods of diagnosis at our disposal; friction cannot always be heard. Friction may possess the usual unmistakable characters, but it may be audible as a dry rhonchus or a moist râle, or be heard to-day and gone to-morrow. Friction is dependent upon the depth of respiration, and it is also modified or annulled by the supine or the erect position. Friction may be heard anywhere over the chest, but the favourite areas are in the neighbourhood of the nipples, in the axillary regions, and about the scapular angles. Friction fremitus also may be detected sometimes, but it is not usually to be felt.

Fluid effusion stage.—The disease may not pass beyond the initial stage, but this is usually followed by that of fluid effusion. Fluid effusion occurs clinically under several types.

In one type, not uncommon, there are the usual physical signs of consolidation of the upper or the upper and middle lobes of the lung, together with deficient vesicular breath-sounds over the lower lobe ; *rarely* are the breath-sounds annulled even at the extreme base. Sometimes the breath-sounds are cavernous over the upper part of the lung, but cavernous breathing is often to be heard over solid areas of a simple nature in children's lungs. Consolidation of the lung may be due to its collapse from fluid pressure or to croupous pneumonia, and if to the latter a trace of albumin may sometimes be detected in the urine in the early stages of the disease. To percussion there is fluid dulness and fluid resistance, but it is important to remember that both of these signs may occur with a solid lung, though this is not usual. On the other hand, because the dulness is not of a character to suggest fluid it does not follow that fluid is not present. The percussion note may be what may be described as deficient resonance, and it is very important to realise that *stomach resonance* may be obtained as high as the angle of the left scapula and thus obscure the physical signs. Percussion should in all cases be light ; the chest walls are yielding, and it is easy to displace the fluid and reach crepitant lung. On the healthy side the breath-sounds are extra-puerile (not bronchial, as sometimes called), and the chest resonance is frequently subtympanitic.

In another type the affected side of the chest will be found to be dull from apex to base, with, in some cases, good but distant vesicular breath-sounds, sometimes with moist râles; or with distant tubular breathing, perhaps heard only on taking a deep breath and with expiration alone. With extreme effusion the apex of the corresponding lung may be resonant or hyper-resonant, the breathing clear but distant, expiration being prolonged and unduly audible in comparison with inspiration. Dulness to percussion does not usually extend to the spine. A sector-shaped area of resonance may be obtained over the root of the lung and its immediate neighbourhood. As in the first type, the breath-sounds on the healthy side are extra-puerile and sometimes accompanied by moist râles.

In a third type there is dulness over the lower lobe only, with deficient vesicular murmur. Sometimes the breath-sounds over the dull area are distantly tubular in character. In either variety there may be loud tubular breathing at the upper limit of dulness, and possibly associated with this friction sounds. Sometimes at this upper limit of dulness friction is heard alone, sometimes pneumonic crackling only. Skodaic resonance is not infrequently to be obtained over the corresponding apex in front, above the clavicle or below it, or over both areas, and sometimes over the apex behind. Skodaic resonance is not diagnostic of fluid; it sometimes is met with in apex consolidation and sometimes with basic consolidation.

In a fourth variety—and this is a very uncommon type—the physical signs are those of pneumo-thorax. I have notes of three cases, and the following is a good example of this condition:

The patient, a hollow-eyed boy, aged 3 years, was admitted into hospital with a two days' history of cough. His respirations were quickened, but he breathed without distress, even when lying down. When disturbed his respiration became grunting, and he resented being interfered with. His chest was resonant back and front when examined in bed in the ordinary way. On the right side, in front, above the nipple, the breathing was vesicular, with just a suspicion of an amphoric character, which latter became more marked as the nipple was approached. Below the nipple the breathing was decidedly amphoric and became increasingly so down to the liver, together with metallic tinkling which was superadded. In front a bruit d'airain was audible in the lower two thirds of the chest, and in the axilla all over the chest. Behind, at the right root, the breathing was bronchial in character, but elsewhere it was vesicular and associated with fine crackling. When laryngeal grunting occurred the grunt terminated in a metallic tinkle, which was audible in the neighbourhood of the angle of the scapula. When the child was shaken, splashing was readily heard, and also a tinkling sound. When he was bent forwards the front of the chest became dull to percussion and amphoric breathing disappeared. When he was bent backwards the back became dull. The heart was displaced to the left, the apex beat being in the fifth interspace, half an inch or more outside the nipple line. The cardiac dulness commenced one and a half finger's breadth to the left of the *left* sternal margin. It was absolute at the fourth left costal cartilage and considerably modified at the third left costal cartilage. Viewed with the X rays, the front of the chest was transparent, except over a triangular strip, with its base downwards, reaching from the third rib to the sternum and liver, which was opaque to the rays, but not so opaque as the heart. This was thought to be the lung. Looked at from behind, the affected side was transparent. The diaphragm on the affected side was distinctly concave above and barely moved with respiration, whereas the other side moved very freely, carrying the stomach with it in its excursions. The chest was opened, and 6 ounces of pus were evacuated, but he died the same night. At the autopsy the heart was much displaced to the left, the right border of the right auricle being about the centre of the sternum. The right lung was collapsed and covered with a thick purulent exudation. A band of adhesion between the middle and upper lobes and the chest wall held the lung in front against the parietes, which divided the pleural cavity into two parts. Pus on the inner side of the lung, between it and the mediastinum, had not been able to flow freely into the rest of the pleural cavity, and several ounces remained there, whereas the part that had been opened had been drained by the operation. Below, the lung was firmly adherent to the diaphragm. At the anterior extremity of the lower lobe three apertures were visible, all of which opened into an abscess cavity. The whole of the lower lobe appeared to be composed of small abscess cavities, apparently broken-down broncho-pneumonia. The upper lobe was almost free from disease, and the left lung only contained a few patches of broncho-pneumonia. Cover-slip preparations from the pus showed a very large coccus, which stained very deeply with methylene blue. It occurred chiefly in small groups of four or five to six, and chiefly as a diplococcus.

Basic consolidation may give all the physical signs of fluid with one exception, and that is displacement of the cardiac impulse. Vocal resonance and vocal fremitus are of but little diagnostic service in children, but cry-fremitus and cry-resonance are sometimes useful. Œgophony as a sign is valueless.

Auscultation of children's chests is by no means easy; the pitfalls for the unwary are numerous. The two sides of the child's chest often do not act in unison: one side works freely, the other is sluggish. Thus it may appear that on the sluggish side there is a deficient vesicular murmur suggestive of fluid, whereas with increased action the breath-sounds become loudly tubular. Thus it often happens that a further examination of the chest coinciding with a fit of crying will materially alter our deductions as to the nature of the complaint which have been arrived at under more peaceful though less reliable conditions. It often happens, too, that at one time a vesicular murmur is audible, whereas with more forcible laryngeal intonation the breathing becomes tubular; possibly the expiratory sound alone has this quality, and broncophony is then heard for the first time. Broncophony contra-indicates fluid.

A case may first come under observation as acute croupous pneumonia, but the customary crisis (sometimes extensive rises and falls of temperature occur after the crisis for a day or two before equilibrium is reached) is passed by, the temperature remains elevated, and the signs of consolidation give way to those of fluid, and this is *the* important point. Sometimes the temperature falls as usual and subsequently rises, remains more or less elevated, and is accompanied by a change in the physical signs suggestive of fluid effusion.

Accumulations of pus in the chest may become localised and, being confined by adhesions, may be limited to the apex, the base, the axillary region, between adjoining lobes, between the lung and the diaphragm, the lung and the pericardium, or the lung and the mediastinum. Thus it may be necessary to explore the chest below the clavicle, or the pus may be quite beyond reach or detection by the methods of physical diagnosis at our disposal. When there are two or more cavities they often communicate by a sinus or by sinuses, but they may be shut off. In a child, aged 10 months, under my care the space containing pus was like a honeycomb.

There is yet another important point, and that is that a deposit of fibrin in the chest may yield all the signs of fluid except that of cardiac displacement.

The position of the apex beat of the heart is a most important aid to diagnosis in pleural effusions. In right-sided effusions the apex beat may be in the nipple line or an inch or more outside this. In left-sided effusions the cardiac impulse may be epigastric, or in the fourth and fifth or the third and fourth interspaces. This impulse is occasioned by the impact of the right ventricle and auricle in the interspaces and of the right and left ventricles in the epigastric region. By some the heart, when it is displaced to the right, is thought to rotate on its axis and by others to be pushed bodily over to the right.

In an autopsy on a child, aged 6 years, the heart was found vertically in the middle line of the body, with the pericardium partially adherent to it. In an autopsy on a child, aged 4 years, more of the heart lay to the left of the sternum, but chiefly behind it. In an autopsy on a child, aged 3 years, the mediastinal contents were shifted to the right, without cardiac rotation. In an autopsy on a child, aged 10 months, with right-sided effusion, the heart was a little displaced to the left. Cardiac bruits are sometimes caused by kinking of the vessels.

The liver and spleen are occasionally displaced by pleural effusions on the corresponding sides of the body. Sometimes the spleen and the liver are enlarged, and the latter may display nutmeg alterations in chronic cases.

The affected side of the chest, which is usually more or less immobile, does not usually display obvious alterations in contour but it may be rounded or even bulging. Sometimes it appears to be smaller. Sucking in of the lower interspaces occurs in some instances. Obliteration or even bulging of the intercostal spaces happens occasionally, and this is best detected by inspecting the chest from above downward. In some cases there is œdema, and increase of cutaneous thickness may be determined by pinching the skin and comparing it with that of the opposite side. Bulging of the chest wall and cutaneous œdema signify purulent collections. In a female, aged 4 years, under my care, with a purulent effusion in the upper part of the chest, this was uniformly bulged forward from the clavicle to just below the nipple, and the sterno-clavicular joint was loosened; the overlying skin was œdematous. In a male, aged 3 months, with a purulent effusion there was marked localised bulging of the lower ribs of the right side.

Empyemas sometimes point externally, usually in one place, rarely in more than one, and mostly to the front of the chest, passing through an interspace, with denuding of the periosteum. These abscesses may be pale or inflamed and painful.

In a boy, aged 7 years, under my care, there was a fluctuating swelling over the front of the left fifth, sixth, and seventh ribs.

In a girl, aged 6 years, the abscess formed over the left fourth, fifth, and sixth ribs.

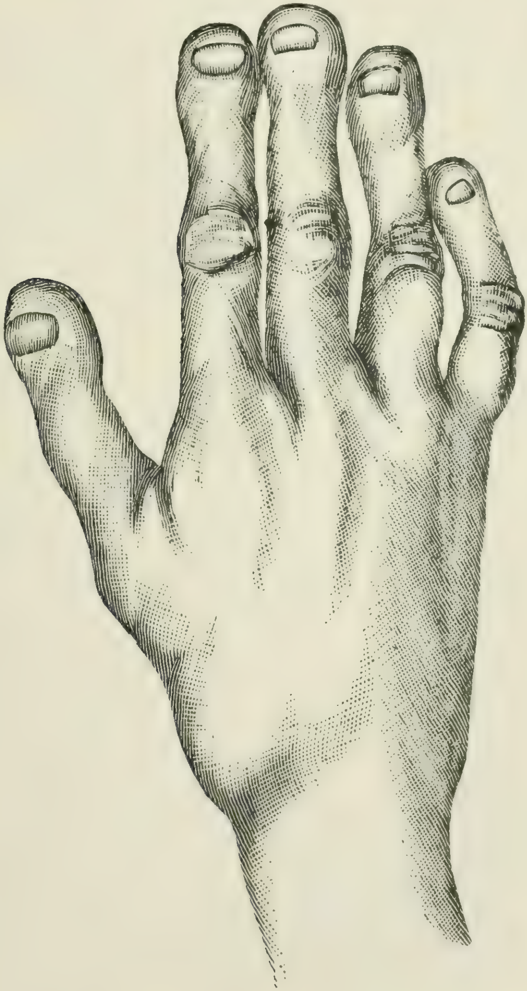
In a boy, aged 3 years, perforation was at the seventh left interspace in the nipple line, with commencing subcutaneous abscess; and a boy, aged 9 years, had an elastic fluctuating prominence over the right hypochondriac region. Sometimes these abscesses have a transmitted cardiac impulse, and the heart-sounds are plainly audible over them. Such are called pulsating empyemas. Pulsation may be detected in the interspaces below and above the nipple, *without* an external tumour. Pus may not only find an exit through the chest wall, but it may track into a bronchus, the abdominal cavity, the abdominal viscera, the loins, or the lower extremities.

The quantity of fluid found in the pleural cavity is variable. The most I have seen was 46 ounces of clear fluid in a child, aged 6 years, but Heyfelder aspirated no less than six pints from a boy of the same age. The smallest quantity I have detected and removed was ½ ounce of pus from each pleura in a child, aged 5 years, with immediate disappearance of physical signs. The average of pus caught at the time of operation is perhaps from 8 to 10 ounces, but matter drains away for some hours afterwards to the extent of several ounces. Aspiration sometimes from admission of air or from instrumental contamination renders serous effusions purulent. I think that an empyema starts as such, but some hold to the contrary. The fluid does alter in character sometimes; thus I have seen at the first aspiration sero-pus, at the second clear blood-stained fluid, and at the third creamy pus. I have seen pus on one side of the chest and serum on the other, and I have also met with a serous effusion on a cured empyema side three years later.

In the early stages of the complaint the child usually lies on the healthy side, but with effusion in quantity he lies on his back or on the affected side, but there are frequent exceptions to this custom. Puffiness of the face and eyelids, like that of renal disease, and pertussis is seen in some cases.

In chronic cases the fingers and toes become clubbed (*vide* Fig. 6), the chest falls in, the ribs crowd together, and the spine is curved laterally, the concavity pointing to the affected side. The child wastes, the right heart hypertrophies and dilates, and the various structural alterations in the organs accompanying this condition make their appearance. Owing to the wasting dropsy is rarely observed. A female, aged 6 years, had been ill two years; the diagnosis

Fig. 6.



was phthisis. She had signs of consolidation in the upper half of the left chest. In the upper half of the chest the breath-sounds were cavernous, below they were either distantly vesicular or distantly tubular. The dulness was absolute. On one or two occasions the opposite root of the lung appeared to be consolidated, but the sounds were found to be conducted, otherwise the breath-sounds were extra-puerile and the resonance was subtympanitic. Expectoration was muco-purulent, sometimes little, at others profuse. She had a contracted chest, with lateral curvature. The cardiac impulse was in the fifth and sixth interspaces, one and a half inches outside the nipple. There was also epigastric pulsation. The free edge of the liver was two and a half fingers' breadth below the costal margin. Her lips were rosy-looking; she was in fair condition and could walk. From the cardiac displacement, the position of the liver, and the physical signs, I thought there must be fluid. Several times I explored, and on each occasion entered solid lung. Finally, I obtained fluid in the axillary region. It was suggested at the time that a dilated pus-containing bronchial tube had been tapped, but 5 ounces of pus were evacuated from the pleural cavity.

Given that there is fluid in the chest, is it simple or purulent? The fact that it accompanies an exanthema, with perhaps diarrhœa, sweating, a sallow complexion, marked wasting, and an obviously low state of health are *in favour* of pus. Temperature as a diagnostic sign between the two is worthless; it may be high with serum, normal with pus. Exploration of the chest by a hypodermic syringe fitted with an exploring needle is the only satisfactory means of discovering the nature of the fluid. Exploration of the chest is a perfectly harmless proceeding if a sterilised needle be passed into a pleura filled with fluid, but it is not unattended by grave risks to life if the lung be wounded under certain and not altogether obvious conditions. It would appear from the published records of fatal cases that wounding a fibroid lung is specially dangerous to life. I was the first to call attention some years ago* to the danger of exploratory puncture of the chest, and since then others have published fatal cases. For a full report of the literature of the subject see the 'British Journal of Children's Diseases,' October, 1905 (editorial, "Exploratory Puncture of the Chest; Abuse of the Needle; A Warning").

When the needle passes into the pleural cavity, fluid, though present in quantity, may not be withdrawn owing to its channel being blocked by fibrin. But the fact that the needle has passed into a cavity can be easily felt by anyone gifted with a delicate sense of touch. So also the fact that the needle has entered crepitant, pneumonic, or fibroid lung will be readily realised. Under the latter circumstances the movements of the impaled lung are imparted to the syringe.

Pus may be obtained from the lung itself by exploratory puncture; thus it may be withdrawn from a dilated pus-containing bronchial tube or from the lung proper. In one case of mine autopsy revealed a pneumonic lung riddled with abscesses, from which puncture almost anywhere would have withdrawn pus.

As has been seen from a perusal of the post-mortem findings of my fatal cases, a certain number of them die from tuberculous affections. The difficulty is to determine during life what cases are tuberculous. The majority owe their origin to simple causes and quickly recover, while others, also simple in nature, do not make the rapid progress that one desires to see, and there is a real difficulty in determining clinically what are tuberculous and what are not. Signs of progressive breaking down of the consolidated lung in the shape of gurgling râles, of fever, wasting, and obvious going downhill betoken phthisis. In the diagnosis of general tuberculosis cerebral symptoms must be watched for. Thus ocular or facial paresis, respiration of irregular rhythm, weakness of a limb, cerebral pulse, sickness not obviously peritonitic, convulsions, and early optic neuritis are important indications of a fatal ending. Suggestive of tuberculosis of the lungs and viscera are—great frequency of the respirations, without sufficient pulmonary cause; a prolonged and irregular temperature, without evidence of imperfect drainage or sepsis; œdema of the hands and feet; and tubercles in the choroid. A tubercular family history and the presence of tubercular lesions elsewhere merit attention. But too great reliance must not be placed on the former, and the presence of other tuberculous lesions is no sure indication of a fatal termination—it warrants suspicion, nothing more.

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* *Vide* 'Reports of the Society for the Study of Disease in Children,' vol. v, pp. 284–506.

Treatment.—For the relief of pain and cough nothing is better than small and frequent doses of opium, administered in the form of compound ipecacuanha powder. Painting the painful area with glycerine of belladonna and the application of warmth to the parts by heated cotton-wool or Gamgee tissue, are useful accessories. Dry cupping is also serviceable, and to a strong and vigorous child three or four leeches may be ordered; but they are apt to produce a bad mental effect in the highly sensitive, and in them should not be tried. Cold applications in the shape of an ice-bag, though sometimes useful, are prone to produce collapse. Hot fomentations, changed hourly, with or without anodynes, have their advocates, but as a rule those topical remedies which require frequent renewal are better avoided. The inoffensive cotton-wool jacket and opium by the mouth usually meet all requirements. If the pain be very severe, increased immobility may be obtained by strapping the chest; the plaster should cross over to the healthy side for a couple of inches back and front. An abdominal binder will help to keep the diaphragm immobile and is sometimes very useful. The child should be put to bed in a room with an equable temperature, say 60° F., and the bowels should be well opened.

When effusion has taken place there are two important considerations, viz. (1) the quality of the fluid, and (2) if serous, the duration of the attack.

If the pleural cavity be full or in sufficient amount to obstruct the heart or respiration, the fluid should be removed *immediately*. Sudden death is very likely to happen in such cases. By quickly opening the chest and performing artificial respiration, I once revived a child who had ceased breathing.

If the lung on the affected side be consolidated, a small amount of fluid may cause considerable cardiac displacement and consequent distress, and the withdrawal of even a small quantity of fluid will confer great relief.

Simple effusions, even if moderate in amount, should not be left *in statu quo* for longer than three weeks, as the lung is liable to contract adhesions. If there be fever it is better not to aspirate, unless otherwise contra-indicated, as the fluid is sure to re-accumulate. When aspirating it is not necessary to remove *all* the fluid: withdrawal of a moderate amount will often suffice. Sometimes two or three aspirations will be found necessary. Aspiration should be stopped immediately the child commences coughing or complains of pain or should blood appear in the bottle. The bottle should not be completely exhausted; it should be filled gradually by a slow pumping action. Should the flow of fluid cease suddenly from blocking of the cannula by lymph, the obstruction can be removed by the introduction of a plunger, smaller but longer than the cannula, and prevented from recurring by keeping it there during the operation.

The majority of cases, however, are amenable to medicinal treatment. A diuretic mixture containing digitalis and iodide of potassium should be given. A useful treatment in some instances is to limit the quantity of fluid to half a pint in the twenty-four hours, and the diet should also be given as dry as possible. Scott's ointment, well rubbed into the affected chest night and morning, or the use of this ointment mixed with iodide of potassium ointment, has seemed to me to be of value. This method of treatment will also be found useful in cases of thickened pleura where resonance is impaired and the breath-sounds are feeble. If pustular dermatitis appears, the application should be temporarily discontinued and the sore places dressed with boracic ointment.

Empyema.—If the effusion be purulent the proper treatment is to remove the pus forthwith. There is but one exception to this rule, and that exception is phthisical empyema because operation in such a condition hastens the tuberculous process in the lung. But even in such a case, if there be danger to life from interference with the heart or respiration, the routine methods of treatment should be adopted. In the cases of empyema that have been under my superintendence nearly all of them have been treated either by simple incision through an intercostal space or by incision combined with the removal of a couple of inches of a rib. I have seen quite a number of cases make a rapid recovery by simple incision alone, but on the whole I prefer incision combined with resection of the rib. When the interspaces are narrow, as is frequently the case, the drainage-tube is very apt to be forced out of the chest by coughing and will be found lying in the dressing. Removal of a piece of rib does not add materially to the severity of the procedure, and its removal allows the introduction of a suitable drainage-tube, it facilitates evacuation of the sodden-looking brown-paper-like masses of lymph which are a feature of infantile pleuritis, and of which I have removed as much as 2 fluid ounces at a dressing, and it enables a digital examination of the abscess cavity to be made at the time of operation. On theoretical grounds it is not advisable perhaps to resect a rib in fœtid empyemas, but even here I have not found the practice harmful. In all the cases under my charge the incision has been made in the eighth interspace just outside the angle of the scapula, and this site has proved in every way satisfactory. It affords good drainage when the patient is lying down and also when he is, as he should be, running about the ward. A rubber drainage-tube about the diameter of the little finger should be selected. It should be sufficiently long to pass well inside the pleural cavity, but not any longer than necessary for that purpose. One end of the tube should be split into three parts; these should be introduced through a hole in a rounded flat piece of rubber and the cut ends bent over and stitched by silver wire to this rubber shield. The tube does not require a hole in it, it does not need to be shortened, when necessary it can be replaced by one of narrower calibre, and when the time arrives to discard it it should be abandoned altogether. When there is much pus it is better on the insertion of the tube to allow the fluid to drain away gradually into a sufficiently thick antiseptic dressing. Serious consequences may follow sudden disturbances of the mechanism of the lungs, such as œdema with frothy expectoration. When the discharge is serous, and but a drachm or so on the dressing, the tube can be discarded. There is no time limit for this; occasionally a week or so I have found sufficient; the average is about ten days. After a few weeks' use of the tube I have several times seen it surrounded by a tunnel of bone, and in some instances several of the ribs have become united by bony growths. The lung can often be seen at the bottom of the wound either of a slate colour or lymph-covered. It may remain motionless or appear close to the chest wall. If close up to the chest, the lung movements appear to be up and down and not to and fro. When there is a large cavity a considerable rush of air will be heard through the tube. Probing the wound, unless there be a large and obvious cavity, is likely to prove misleading. Even if there be *apparently* a cavity of some extent, the discharge being serous and trifling, an attempt should be made to abolish the tube. After operation usually the air appears to enter the chest fairly, with vesicular breath-sounds. But stethoscopic signs are somewhat misleading. Thus I have heard vesicular breath-sounds over a completely collapsed and therefore airless lung.

When the upper lobe is consolidated the physical signs may persist for a long period. I have noticed this with even a moderate-sized empyema. Thus, in a child, aged

2 years, with left-sided empyema consolidation of the upper lobe persisted for two months, and then rapidly cleared. In another child under 2 years similar consolidation continued for eight months after the empyema was cured. Mercurial inunctions were tried. Two months later there were signs of resolution, resonance returned, breathing was vesicular, entry of air fair, together with a few râles. When he next came under observation the chest was quite healthy, and, with the exception of scarring, no difference could be detected from the opposite side. Recognition of these cases of delayed resolution will prevent a too hasty diagnosis of phthisis. Below the level of the wound may be heard distant tubular breathing, a vesicular murmur, or râles. When the empyema is cured there is apt to remain deficient entry of air and impaired resonance for some little time to come.

With re-accumulation of pus there are two indications, viz. a rise of temperature, sometimes not much above 99° F., and alteration in the physical signs. A rise of temperature does not necessarily mean imperfect drainage or re-accumulation of pus. In several of my cases it was due to scarlet fever or measles and in others to pneumonia, tonsillitis, general tuberculosis, or peritonitis. Sometimes it could not be accounted for, and, again, it appeared to be owing to a small superficial collection of pus about the wound. Conversely a subnormal temperature need not cause anxiety; this is very common in childhood, and not infrequently the temperature may be as low as 95° F. A re-accumulation is not always easy to diagnose. Thus a child, aged 6 years, a cured empyema, was suspected of typhoid fever in the pre-Vidal days. There was comparative dulness and somewhat deficient breathing, quite compatible with her history. Three times she was most carefully examined. After fifteen days a swelling appeared over the scar and a quantity of pus escaped.

It must be admitted that a certain number of cases of empyema in children are never diagnosed, and recover spontaneously. It is also undoubted that simple aspiration, usually several times repeated, is capable of curing empyema. Small collections of pus are sometimes expeditiously dealt with by this method. Thus pus was withdrawn by exploratory puncture from the right base. Some days afterwards, when the child was prepared for operation, the physical signs were rather in favour of solid lung. Several exploratory punctures were made and passed into solid lung. The chest signs finally cleared. The quantity of pus in this case was probably small. But time is certainly not usually saved by the use of this method, and repeated aspirations cause far more constitutional disturbances than one simple empyema operation. Further, the method has the disadvantage of leaving flakes of lymph behind in the pleural cavity, which possibly may set up mischief later on. Aspiration, therefore, cannot be recommended.

If the empyema has ruptured into a bronchus, then if the discharge be diminishing, if the child be gaining flesh, and if the temperature be normal, the cure may be left to Nature. But if the child be not so fortunately circumstanced, then an incision should be made into the pleural cavity and the empyema treated in the ordinary way. When this is done, the opening in the bronchus, if small, often rapidly closes; occasionally, however, the track in the lung does not heal, and such cases are very unpromising. If the empyema be pointing externally, the chest should be opened at the site recommended for the treatment of ordinary cases, unless the collection of pus be local, when it should be drained at the most dependent situation. If that be where the abscess is pointing, then the wound must be enlarged, but if not a second opening should be made in the most favourable place.

When a child has worn a drainage-tube for months and the discharge continues purulent, when the lung has expanded to its utmost, and when the chest walls have fallen in as far as they will go, and the diaphragm has risen as far as it can rise, and there still remains a cavity, it is advisable, following the plan of Estlander, to facilitate the fall of the chest upon the lung by removing large portions of ribs bounding the cavity. The largest portions should be removed opposite the middle of the cavity and the smallest opposite the upper and lower ends. The whole of the periosteum and thickened pleura corresponding to these ribs must be excised to prevent the re-formation of bone. Symptoms of lardaceous disease make the closure of the cavity imperative; and in chronic cases, where the chance of expansion is problematical, it is best to resort at once to resection. Thus a child on each occasion healed after twice undergoing ordinary surgical treatment for empyema and finally underwent successfully resection of ribs. The disease in this case lasted over a period of five years. She was seen some long time afterwards, and the chest, with the exception of scarring, showed little, if any, trace of the empyema.

An anæmic, wasted girl, aged 4 years, with a history of illness of some months' duration and with a shrunken chest, slight lateral curvature, a puffy-looking face, and a strong family history of phthisis was admitted into hospital with a left-sided empyema which had pointed just below the corresponding nipple. The heart was displaced to the right. The empyema was opened in the eighth interspace, just outside the angle of the scapula, and 5 ounces of pus were evacuated. Between the shrunken lung and the chest wall there was quite a large cavity, and subsequently pieces of five ribs were removed, four inches in all. Two months later the wound had healed, but the heart still remained under the sternum, the breath-sounds, however, had improved all over the left side, and the child left the hospital in good condition.

Sinuses are troublesome in some few instances. In one child under my care many inches of rib were exfoliated. Occasionally resection may be necessary to bring about their closure. Washing out the pleural cavity serves no good purpose, and is dangerous. Death from this practice has occurred from coma, convulsions, and hyperpyrexia. If the pus be stinking, washing out may be tried, but I have never seen any good results from it.

Finally, there are two *most important* points in the after-treatment of empyema, viz. plenty of fresh air and exercise short of fatigue. The child should not be kept in bed longer than is absolutely necessary, and immediately his strength permits he should be up and walking, and finally encouraged to run about in the open. Get the empyema patient out of bed is a golden rule of practice.

(*Read December the* 15*th,* 1905.)

37. PLEURAL EFFUSIONS, SEROUS AND PURULENT: APPARATUS TO ILLUSTRATE IMPORTANCE OF FREE OPENING FOR EMPYEMATA.

By ARTHUR EDMUNDS, M.S.

Mr. Arthur Edmunds said he desired to exhibit an apparatus to demonstrate the importance, in relieving empyema, of making the opening into the chest sufficiently free, not only to allow of the pus being forced out voluntarily, but for other reasons. Although everyone has realised that the chest did fill in coughing and in violent expiration, he did not think it had been sufficiently realised that that method of expiratory filling of the affected lung was one which was constantly taking place. When the lung on the other side was connected with the affected one by a bronchus, and when the passage outwards was free, as in paralysis of the larynx, the movements of the chest-wall would have no effect on the movements of the other side; alterations in the tracheal pressure were not able to affect that lung. But if there was an obstruction in the outflow from that tube which took place by the movements of the vocal cords, then on expiration the unsupported lung where there was an opening into the chest-wall would move also. That, he believed, took place not only in violent coughing, because he had shown by experiments on animals that after opening the chest-wall and setting up an irritative reflex from the pleural cavity the examination of the vocal cord was sufficient to cause blockage, so that while the lung on the sound side was collapsing the lung on the other side was expanding. That was the process which he felt sure was going on during the whole course of healing of an empyema. (*Exhibited December the* 15*th*, 1905.)

38. CASE OF LEFT PLEURISY WITH EFFUSION; PARACENTESIS BY SYPHONAGE WITH ADMISSION OF AIR; SUBSEQUENT RELAPSE OF THE EFFUSION TREATED BY INTRA-SEROUS INJECTIONS OF ADRENALIN WITHOUT PARACENTESIS; RECOVERY.

By WILLIAM EWART, M.D.

The patient, a boy, aged 10½ years, was relieved of his effusion on the seventh day of his attack. He had some dyspnœa with slight cyanosis and displacement of the heart to the right. Some crepitations and impaired resonance were noted at the left apex. The temperature was 102° F. and the respirations 48 per minute. The fluid proved to be serous, and was removed under slight anæsthesia by syphonage through a cannula introduced at the seventh intercostal space in the anterior axillary line. A second cannula was introduced at the third interspace, and connected with an air supply derived from a Wolff's bottle and filtered by passage through cotton-wool. The air was allowed to find its way into or out of the chest without any external pressure, but alone under the influence of the intra-thoracic tensions as they prevailed at each moment. In short, there was no injection but only admission of air. The result was a smaller air intake (477 c.c.) than fluid outflow (52 ounces), and no such alteration in the intra-thoracic pressure as to lead to a return of the heart to its normal position.

The dyspnœa was much relieved in spite of the artificial pneumothorax, which lasted until June the 26th. Though doing otherwise quite well, the patient re-accumulated fluid as proved by the return of Grocco's triangle and of splashing. The fluid dulness and Grocco's triangle continued to rise (until June the 22nd), up to the level of the fourth spine, but the heart had only partly moved back to the left.

This secondary effusion was dealt with successfully without any other treatment than the intra-thoracic injection of hydrochloride of Adrenalin's solution ($\frac{1}{1000}$). Of this 10 minims were injected on June the 17th into the serous accumulation, with the precaution of identifying the latter by drawing a few drops into the half-charged hypodermic syringe before discharging its contents into the pleura. The injection was repeated on June the 20th, June the 22nd, June the 26th, and June the 28th. After the first injection the dulness continued to rise in the back; but the heart began to return towards the left, showing improved pulmonary expansion. After the second injection the dulness ceased to rise, the breathing improved, and the heart gradually returned to the normal site.

After the third injection the dulness and Grocco's triangle, which had already diminished, rapidly dropped to a low level; and on June the 28th, when the last injection was made, Grocco's triangle was noted to be absent, and only slight impairment of resonance remained at the base. Subsequently the chest was found to be quite normal, and the patient has remained in excellent health.

(*Read December the* 15*th*, 1905.)

39. A CASE OF CUTANEOUS PIGMENTATION.

By ARTHUR EDMUNDS, M.S.

The case exhibited shows on the buttocks and back a curious bluish mottling similar to the pigmentation which occurs in a large number of Asiatic races of mixed descent. The pigmentation resembles that shown in a Japanese baby recently exhibited at the Society by Dr. Sutherland.

Much discussion has been caused by this pigmentation, some holding that it is an evidence of a negroid ancestry, while others, especially the Japanese authors, hold that it is merely a racial characteristic. In the present case there is no history of any negroid ancestry.

The lower animals exhibit a great variation of pigmentation, and in a very large number of species examples of albinism and of melanism are reported, and it seems unlikely that men should be exempt from this type of variation. Among the lower animals it seems to be entirely erratic, and if it occurs in man one would expect to find the same characteristics to hold.

Albinism is well known in man, with the pink eyes and extremely fair hair, and there does not seem any reason why one should not regard a case like the present as one of melanism. The distribution of the pigmentation is exactly what one would expect. The dorsal region is nearly always more deeply pigmented than the ventral, and this obtains in the present case.

The pigmentation was noticed during the examination of the child, who was brought for the treatment of rickets.

(*Exhibited January the* 19*th*, 1906.)

Discussion.

Dr. G. A. Sutherland said he felt very much interested in the case, because until he met with the Japanese baby which he showed before the Society a short time ago he had never observed a similar pigmentation. He had recently received a letter from Dr. Ashby, of Manchester, stating that that gentleman had an English baby brought to him with blue coloration of the skin, similar to that of the present case. It was curious that men of such large experience as Dr. Ashby had never met with the condition previously, and yet three months after the reference in the 'British Journal of Children Diseases' three cases were reported to the Society. Probably such cases had previously been overlooked, no doubt because the impression conveyed at a glance was that there was a sort of superficial nævoid condition, but not sufficient to call for the active interference of the surgeon. No doubt, as Mr. Edmunds suggested, it was a condition of melanosis in the Japanese baby; various pigment cells had been discovered in the superficial and deep layers of the dermis. But it was difficult to explain the occurrence in European children. Perhaps in the Japanese it might be traceable to a blacker skin in a previous race. With regard to the occurrence in Europe of the condition, his attention had been directed to it by a contribution by a Japanese doctor in Germany, the title being "On the occurrence of the blue pigmentation of the Japanese in German children." He discovered several cases of the kind in a German clinique; and that was a confirmation of the point mentioned by Mr. Edmunds, that the Japanese condition might occur in white children.

Mr. George Pernet said no doubt the pigmentation in the present baby was absolutely the same as that in the Japanese infant shown by Dr. Sutherland recently. Mr. Edmunds was at liberty to call it "melanism" if he liked. As to melanodermia, that did not quite apply in the present case, dermatologically speaking. He (Mr. Pernet) thought it would be difficult to escape the ancestral factor. The family history of the child had not been investigated, and the father had not been seen. In this case the possibility of such a factor could not be left out of the biological purview. It might be found that, as Dr. Ashmead of New York averred, it was really a negro mark. He (Mr. Pernet) had seen the skin of hundreds of children, but had never observed the condition in a white child before as far as he could recollect.

40. A CASE OF SCLEREMA NEONATORUM.

By GEORGE CARPENTER, M.D.

Mary B—, aged 8 weeks, was admitted into the North-Eastern Hospital for Children on December the 30th, 1905, for a peculiar condition of the skin which was said to have existed from birth. The labour was difficult and artificial respiration had to be resorted to, but since then the child has been vigorous and has gained weight, and cried lustily. This child is the last of seven, and the others were all healthy.

The infant is well nourished, takes food well, weighs 9 lbs., the colour of the skin is good, there is no cyanosis, and the temperature is normal. There is a brawny induration of a pinkish-purple colour and of a less even outline than usual, having some well-marked dimples and rounded, but slight eminences, which extends over the whole of the back, and back of the neck, buttocks, and upper part of the thighs at the back, and then tapers to a point just above the back of the knee in a very characteristic way, and also spreads over the deltoid region and posterior part of the axillæ. Two indurated patches are present on the occipital region of the scalp. These areas do not pit on pressure, but the pinkish colour fades, and the hyperæmia returns slowly, when the skin is pinched up between the finger and thumb, and the underlying tissue being closely adherent appears when doubled together to keep the fingers more than one inch apart, and gives a sensation as if a layer of indiarubber were adherent to the under-surface of the skin. The skin of the front of the body is not affected. The general condition of the child is satisfactory, the temperature being mostly normal, but on one occasion it rose to 103° F., without any obvious reason. Pulse rate 160 to the minute on admission, but lately it has been about 130; well felt at the wrist. The respirations are of normal rate, and the lungs appear healthy. The bowels act normally, and the motions are well digested. There has constantly been a trace of albumin in the urine. The child has gained $1\frac{1}{4}$ lbs. in a fortnight, its weight now being $10\frac{1}{4}$ lbs.

The mother states that the hardening of the skin at present is exactly the same as it was at birth, and has not altered in any way. (*Exhibited January the* 19*th*, 1906.)

Postscript, October, 1906. — Under the heading of "Microscopical and Chemical Observations on a Case of Sclerema Neonatorum," the subsequent history of this patient, together with the under-mentioned observations, were published by Dr. George Carpenter and Mr. Sheffield Neave in the 'Lancet' of July the 21st, 1906. The following account is taken *verbatim* from that journal.

Later these extraneous signs extended slowly and to a less degree to the angles of the jaws, the sides of the neck, down the arms nearly to the wrist, and there were islets formed all over the abdomen and chest. Pain was never an accompaniment of these indurations. During the five months the child has been under observation, although the above extensions took place, these signs have cleared up irregularly but continuously. At the time of writing a small triangular patch of induration exists between the shoulders with the apex downwards on the spine, but it is not nearly so thick or hard as in the first place. There are a small patch on the right buttock and an islet on the right cheek just below the superior maxilla. The glands are somewhat enlarged and shotty in the posterior triangle of the neck, and in the groin and the spleen reaches one finger's breath below the ribs. Throughout there was nothing distinctive taking place in respect of the temperature. A blood-count was made but gave nothing very definite—viz. polymorphs 34, lymphocytes 57·5, large mononuclears 0·6, myelocytes 0·5, and eosinophiles 2, total 100, erythrocytes 4,212,000, and leucocytes 22,500. The child has during her stay in the hospital thrived very satisfactorily and the skin has cleared up as described under massage, together with, first, thyroid and then thymus extract given internally. The thyroid was first given for a month, when some improvement was made. Thymus was then given, beginning with a small dose and being raised to 30 grains *per diem*, though subsequently reduced to half that amount. During this last treatment progress appeared to be much hastened.

A small piece of skin and subcutaneous tissue down to the muscle was excised by Mr. J. P. Lockhart Mummery from the back of the thigh for microscopical examination. Macroscopically, the fat and superficial fascia appeared to be perhaps thicker than usual but only slightly. The fat, however, was certainly whiter and in larger globules than usual and was much harder to the touch. Nothing further abnormal was, however, detected under the microscope, a control examination being made at the same time of a similar specimen of healthy skin from another child of the same age. The serous infiltration and increase of connective tissue mentioned in some cases were certainly absent and in this way this case agrees with that of Northrup mentioned by Holt.

The fat was submitted to Professor J. Addyman Gardner, M.A., F.I.C., for examination. He reported as follows:

"I am afraid my investigations do not throw much light on the case. There are two difficulties which it was impossible to get over: (1) the small quantity of the sample, and (2) the fact that it was probably somewhat altered by the prolonged soaking in the formalin fluid. The dry specimen contained only 58 per cent. of fat, the rest being ordinary tissue matter, as far as could be determined in the dried residue after extraction, and this appears to agree with your microscopical report. The saponification equivalent was 205. The specimen also contained unsaturated fat—*e. g.* olein—but it was too small to permit of a quantitative estimation. The values usually given for the saponification equivalents of the various pure fats are: tripalmitin 209, tristearin 189, triolein 190, horse fat 190·4. One might conclude from this that the specimen was particularly rich in palmitin. In the hope of throwing some light on the matter, I examined the saponification equivalents of specimens of human fat cut from various parts of the body but they showed very considerable variation.

"I thought at first that the specimen of fat and the acid from it seemed more solid than is usual in human fat, but my examination of the specimen mentioned scarcely bears this out.

"On the whole, I am inclined to doubt whether the fat in the sample was in any way abnormal, and think that the cause of the trouble must be sought in the associated matter."

It is thus obvious that the pathology of the disease is very obscure and it is hoped to call attention to the malady by the publication of this case, so that the work done here, although so negative, may be of some use to others as a starting-point for further investigation when new cases are obtainable.

41. A CASE OF SCLEREMA NEONATORUM.

By J. PORTER PARKINSON, M.D.

Benjamin S—, aged 1 week, was brought to the North-Eastern Hospital for Children on November the 28th last year, on account of lumps in the skin and inability to take food. The patient was recognised to be suffering from sclerema neonatorum, and was admitted into the hospital. He was an eight months' infant, the mother having had two other children previously, both of whom are alive and healthy. She had had no miscarriages. The father and mother, who belong to the Jewish race, both seem healthy. There is a strong family history of phthisis, the mother's mother having lost eight children from this cause.

The condition on admission was as follows. The infant was obviously very feeble, whining slightly at times, and being unable to cry properly. Over both cheeks, nose, and eyelids there was a brawny induration distinctly elevated above the surrounding skin, which was of a reddish-yellow colour, not differing from that over the rest of the body. The indurated area did not pit on pressure, and had a hardish feel, which the house-physician compared to that of a ripening peach. Similar indurated elevated areas were found more or less symmetrically over both clavicles, over the spines of the scapulæ, and a small area over each buttock, On introducing the finger into the mouth the tongue felt hard, and the child did not suck. The temperature on admission was 97° F., but fell later in the day to 96° F. The rate of breathing varied greatly between 32 and 64 at different times. The pulse rate was 136. The breath-sounds were weak, but of a harsh quality over the lungs, and there were no abnormal signs in the chest or abdomen. The infant weighed 4 lbs. 12 oz.

In the absence of an incubator, the child was kept as warm as possible in a cot by means of hot bottles, and as it was totally unable to suck it was nasally fed. The first attempt at this brought on stoppage of the respiration with cyanosis, and the child was saved from immediate death by Schultz's method of artificial respiration. These attacks of asphyxia recurred from time to time. Some hours after admission a vesicle appeared on the bridge of the nose and another on the right eyelid, which increased rapidly to the size of peas, being filled with a turbid brown fluid which eventually became black; the vesicles then burst, leaving small ulcers. The areas of induration remained as on admission, but the child died two days after it was first seen from asphyxia, the temperature having been subnormal during the whole time it was under observation. A few hours before death the scalp was found to be œdematous, pitting deeply on pressure, but there was no other œdema present. At the necropsy there was nothing abnormal found beyond congestion of the bases of both lungs, with patches of collapse at the apices. A portion of the indurated skin over one of the scapulæ was excised and examined microscopically by Dr. Sydney Curl, who was at that time pathologist to the hospital. The following is his report:

"(1) The epidermis appears to be considerably reduced in thickness, and the transition from the Malpighian cells to the corneous layers is less gradual than normal, and degeneration of the most superficial corneous layers is well marked.

"(2) The papillæ of the true skin are badly marked, and in some parts of the sections are entirely absent.

"(3) The cutis vera, which appears much thickened, is composed of bundles of fibrous tissue, the upper layers of which contain numerous connective-tissue cells; in the lower layers these are fewer in number. Mingled amongst these fibrous strands are numerous blood-vessels, some of which are very large, hair-follicles with sebaceous glands, and sweat glands with their ducts. A few masses of fat cells are also to be seen here. Beneath the true skin is the ordinary fatty subcutaneous tissue."

In these microscopic specimens shown, the appearances of a portion of skin excised from my case is compared with a specimen of normal skin taken from the same region of the body of a child who died from another disease. It will, I think, be allowed that the two specimens show no marked differences, illustrating the view that the process is rather a chemical than a structural one.

(*Exhibited January the* 19*th*, 1906.)

Discussion.

Mr. George Pernet said Dr. Carpenter's case was certainly one of partial sclerema neonatorum; it was of rare occurrence, and was more likely to be seen in a general children's clinic than in a skin clinic. Generalised cases were rarer still. The temperature in the present case was normal instead of being subnormal, and it was interesting to note that there was considerable trouble in keeping the child alive at birth. It was true, as Dr. Parkinson said, that the section of skin in his case did not go deep enough to show the condition below the corium. Kaposi had compared the condition as analogous to stearin formation. According to Langer, the fat of the new-born solidified at a higher temperature than did the fat in adults.

42. A CASE OF LIPOMATOSIS.

By LEONARD GUTHRIE, M.D.

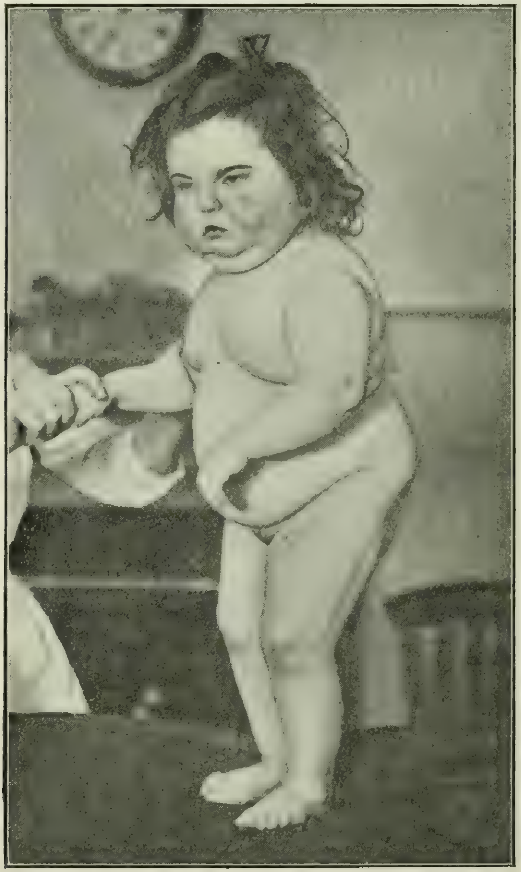
E. W—, female. Always rather plump, but increasing obesity noticed for three or four months. Brought to hospital on this account, and also because the child had "no life in her," was always wanting to sleep, complained of pain somewhere in head or face, and frequently became blue and cold.

Present condition.—A very fat child. Height, 2 ft. 8½ in.; weight (December the 7th), 32¼ lbs. Neither weight nor height abnormal. Discrepancy between weight and appearance probably due to lightness of skeleton. Adiposity is general, but also distributed in soft pads or rolls round neck, over lower cervical and upper dorsal vertebræ, in posterior folds of axillæ, and in ilio-inguinal region, but not above clavicles. *Thyroid gland* not felt. *Cardio-vascular system:* circulation poor; extremities cold. General congestion of face and lips, not amounting to cyanosis. *Heart's* action rapid, irregular, and intermittent. First sound weak with occasional systolic bruit at apex. Second sound accentuated. Pulse averages 120 to 140 beats a minute. *Lungs* normal. *Urine* alkaline; specific gravity 1020; no albumin; phosphates abundant.

General appearance not cretinoid. Hair is soft and abundant, skin moist and supple. Eyelids not puffy; lips not thickened nor mauve-coloured. Tongue neither large nor protruded. Movements are not deliberate and lethargic. Child is not placid and apathetic, but passionate, sensitive, exacting attention, and mentally bright.

The obesity seems a morbid condition, not a mere "normal abnormality" (Goodhart). Possibly there is a connection between deficient thyroid development and lipomatosis, whilst in the latter lessened oxidation owing to the cardiac condition may play a part. It is suggested that the case may be one of arrested development with obesity ('Nanisme avec Obésité,' Bourneville).

FIG. 7.



Child is under thyroid treatment at present. Loss of weight in three weeks equals $1\frac{3}{4}$ lbs.

(*Exhibited January the* 19*th*, 1906.)

Postscript, October, 1906.—Three months later the child developed signs of precocious puberty. At present (September, 1906) there is a thick growth of hair on the pubes, the mammæ have become large and pendulous, and the general obesity has increased until the patient can hardly walk. There have been no signs of menstruation.

The case is probably, as Dr. Parkes Weber suggested on January the 19th, one of precocious sexual development associated with adrenal tumour (hypernephroma). Such cases in females seem attended by abnormal obesity, in males by excessive muscularity, strength, and height approaching virile type.*†

Discussion.

Dr. F. Parkes Weber drew attention to the resemblance of the present case to one shown by Dr. Still in 1901 at the Clinical Society, the record being in Vol. xxxiv, p. 243, of that Society's 'Transactions.' At the end of that report Dr. Still said: "The heart shows nothing abnormal except an occasional slight irregularity of rhythm and marked accentuation of the second sound in the aortic area." Dr. Still afterwards told him (Dr. Weber) that the progress was not satisfactory, and death occurred with cerebral symptoms, possibly indicating cerebral hæmorrhage. He believed the outlook was bad in Dr. Guthrie's case. Dr. Weber suggested that the precocious plethoric obesity in the child shown by Dr. Guthrie was possibly due to the presence of a hypernephroma either in the cortex of the suprarenal capsules or in one of the other abdominal viscera—*i. e.* a tumour derived from cells of the kind found in the cortex of the suprarenals. This supposition would account for the cardiac symptoms. He did not know whether Dr. Guthrie had examined the blood-pressure, but that probably was not necessarily excessive in hypernephroma cases. Slowly-growing hypernephromata might give rise to unusual growth in children and to congestion of the face and cyanosis (*vide* White and Nisbet, 'Arch. de Pediat.,' 1904, p. 943).

Mr. Milner Burgess (Harlesden) said he had a case of the kind under discussion from infancy, the girl now being thirteen years of age. She recently passed in Honours at the Oxford Local Examinations. She was so fat that he would have liked to have shown her at the Society, but the parents would not consent. She had been taking thyroid extract, with very good results. In reply to Dr. Weber, he said there were no cardiac signs and no plethora.

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* *Cf.* Bulloch and Sequeira ("Relations of Supra-renal and Sex Organs," 'Path. Soc. Trans.,' 1902).

† *Vide* "A Case of Precocious Development," by Hugh Lett, Ch.B., pp. 200–03.—[Ed.]

Dr. G. A. Sutherland said Dr. Guthrie had raised some very interesting points about the child. The ordinary observer was struck by the adiposity. Superficial examination did not reveal anything else very marked. The class of case to which Dr. Guthrie had referred, namely nanism with obesity, was fairly common, and one could usually discover something besides the mere excess of fat. He did not think there was any particular mental backwardness in the present child; indeed, it was rather precociously sharp, and it had a command of language which was more striking than commendable. Dr. Guthrie had referred to cretinism and myxœdema, but so far as he (Dr. Sutherland) could discover, the fat was diffused generally all over the body. Before going in for refinements of diagnosis or treatment, one would like to know what the child had been fed on. He would like to try the effect of cutting off the carbohydrates and fats, and in that way getting the weight down. He had seen the child eating freely of bread and butter, and while that continued he did not think there would be much reduction by thyroid treatment. The condition of the heart had been dwelt upon, and the heart in the present instance he understood to be rapid, irregular, and weak, and there was a tendency to cyanosis. In a child as fat as the present one the heart would naturally have great difficulty in carrying on the circulation because of the enormous capillary obstruction. If the weight were reduced 6 to 8 lbs. he would not be surprised if the circulatory condition were much improved.

Mr. N. Bishop Harman did not regard the condition as a very common one. He examined in one year 7000 children in the Hackney Board Schools and saw only two with that abnormal fatness. There seemed to be nothing pathological about them except their enormous bulk; they could do their physical drill as well as the others, though they sweated more profusely from it. There was no great cardiac distress, and the intelligence was rather above than below the average. In a girl, aged 13 years, who would easily have passed for eighteen, there was a great deal of fat, and there was some precocity. This adiposity was sometimes observable in families. He knew a family of girls all of whom except one were very thin, and the exception was very fat. In another family one child of six years weighed 5 stone 2 lbs., and the older sister of ten years weighed 5 stone 2 lbs. Both were healthy and strong. One child of another family was so fat that she was named "Roley Poley," but in later years she was very thin. There must be some reason for it, but nothing has been discovered to adequately explain it.

Dr. Leonard Guthrie, in reply, said he had not suspected the possibility of there being any suprarenal disease, but it was worth bearing in mind. He agreed with Dr. Sutherland that there was no mental backwardness in the child, and that its language was capable of improvement. Bourneville's cases were certainly associated with mental enfeeblement, though he did not see why that should necessarily be so in every case. Still, taking into consideration the possibility of deficient thyroid development, one would imagine the whole body and mind to be deficient. Perhaps if the child ceased to grow the mind might also cease to develop. He could not agree that the question of carbohydrate food entered largely into the case, because the condition had been increasing during the last few months and the child had been living in very poor circumstances and was not at all likely to have been over-fed any more than it was, whilst under treatment. He was unable to agree with the statement that the fat was evenly distributed throughout the body; it occurred in pads between the shoulders, and that was the distribution which one met with in cases of thyroid deficiency. Still, as he said, he did not wish to be dogmatic about it, and would be pleased to show it at a future meeting.

43. INFANTILE HEMIPLEGIA WITH ALMOST COMPLETE RECOVERY.

By C. O. HAWTHORNE, M.D.

The patient was a boy, aged 8 years, who when two years of age had a severe convulsive seizure followed by paralysis of the limbs on the right side.

He was presented to the Society as illustrating the extent to which recovery is possible in this class of cases. The only present evidences of the hemiplegia are some relative weakness in the right grasp, clumsiness in the more delicate movements of the right hand, exaggerated knee-jerk, and extensor plantar response in the right lower limb. There are, however, other indications of damage to the nerve centres in the shape of some degree of mental deficiency and occasional quasi-epileptic seizures. Patient is the youngest of thirteen children (eight living); there is no evidence of specific taint. The immediate cause of the hemiplegia was probably an acute encephalitis.

(*Exhibited January the* 19*th*, 1906.)

Discussion.

The Chairman (Dr. Porter Parkinson) said Dr. Hawthorne's case reminded him of one which he showed at the

Society about a year ago—that of a child who recovered from a passing attack of hemiplegia. It had permanent weakness of the right side of the face, the paralysis of the limbs being on the left side. The latter paralysis passed away in a few days. He took it to be a case of polio-encephalitis inferior, and that Dr. Hawthorne's case must be of a somewhat similar kind. He did not understand whether the recovery of the use of the limbs in Dr. Hawthorne's case had been fairly rapid or only very gradual.

Dr. C. O. Hawthorne, in reply, said he was obliged to depend on the statements of the mother on the point, as he did not see the child in the attack, but only during the last eighteen months. According to the mother's story the limbs remained completely paralysed for some months, and since then had gradually improved.

44. TWO CASES OF MENTAL DEFECT ASSOCIATED WITH OPTIC NERVE ATROPHY.

By C. O. HAWTHORNE, M.D.

These cases were presented as examples of early degenerative brain disease in association with optic nerve atrophy. The clinical summaries are as follows:

(1) Edna C—, aged 7 years, brought to hospital with the complaint that she is "very nervous, easily frightened and can't learn anything." Patient is obviously mentally deficient. She is reported to be generally good tempered, and clean in her habits, but liable to outbursts of temper; at times she "sees" persons who are not present. Vision is certainly defective and the optic discs are very white and clean-cut; there are no other changes in either fundus. The pupils act to light and in accommodation; there is manifest nystagmus, in part vertical. Irides greyish-blue; hair very fair. Patient is well grown for her age, and is physically quite strong and well. There are no evidences of hereditary syphilis. The tendon jerks are normal; there is no muscular tremor.

The paternal grandmother is an inmate of a lunatic asylum, but there are no other evidences of family nervous disease. Patient is the fourth of five children; the others are described as "very sharp." There was an interval of five years between the birth of the third child and that of the patient, but neither then nor at any other time is there a history of a miscarriage. Patient was born without difficulty; there is no history of injury or of serious illness.

(2) Ernest C—, aged 12 years. Possibly never "quite right," but went to ordinary school until seven years of age and there reached standard ii; since his seventh year his mental condition has gradually deteriorated, and now "he cannot be taught anything"; recently he has had a number of fits.

Patient is childish in manner and incapable of sustained attention; at times he has outbursts of temper; his habits are reported to be cleanly; no history of delusions.

The pupils are of medium size; they have no light response, but contract freely during convergence. The eyeballs show almost continuous coarse rolling movements, and there is also a fine nystagmus. Vision is very defective; the optic discs are very white, and there are fine choroido-retinal changes with, in the extreme nasal periphery, a few small areas having the characters of a disseminated choroiditis. Irides brown in colour; hair of scalp blackish.

Patient is rather tall for his age, and his muscular development is fair; the testicles are not present in the scrotum. There is no tremor. The knee-jerks can hardly be obtained. The frontal eminences are somewhat prominent, but otherwise there is nothing in the physiognomy to suggest a specific taint.

There is no family history of nervous disease. The mother has had four pregnancies; the first child was born at the seventh month and died at birth; the second died at six years of "inflammation of the bowels"; the present patient is the issue of the third pregnancy and was born at full time; six years later another boy was born, and is now physically and mentally a vigorous and robust child without any evidence of specific taint.

In each of the above cases the gradual progress of the mental deterioration and the absence of any clinical event capable of being regarded as a signal of the occurrence of a gross organic lesion, seem to compel the admission of a process of slow degeneration, presumably in the cerebral cortex. Such a conclusion receives further support from the coincident optic nerve atrophy. Hence it would seem proper to place these cases among the group which Sir Wm. Gowers has named *abiotrophies*.* They thus come into relationship with certain forms of spastic paralysis seen in early life, and due to degeneration of the upper motor neurons. Another possible relationship is with juvenile general paralysis. In this latter connection it may be noted that the boy (case 2) is not free from the suspicion of congenital syphilis, and that the pupils have no light response and the knee-jerks are defective. Whether specific disease is or is not responsible does not seriously affect the view which is suggested in reference to the essential nature of the pathological process in the nervoûs system, though in the one alternative the degeneration will be attributed to a definite toxin and the other to an unknown influence which may be named "a defect of vital endurance." The chief interest of the cases seems to be the force which is lent to the presumption of degeneration in the higher reaches of the nervous system by the accompanying fact of optic nerve atrophy. That in each instance the child was born after the occurrence of several years' freedom from child-bearing is perhaps no more than a coincidence. Presumably the same explanation applies to the fact that each child has the same surname, though the families have no knowledge of each other's existence. It is noteworthy that so far as can be ascertained the other members of the same generation in each family are free from mental defect, but isolated examples in the earlier years of life, both of unexplained nerve degeneration and of the effects of inherited syphilis, are, of course, not uncommon.

(*Exhibited January the* 19*th*, 1906.)

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* The 'Lancet,' April the 12th, 1902.

45. A CASE OF HEMIHYPERTROPHY.

By GEORGE CARPENTER, M.D.,
AND
LOCKHART MUMMERY, B.C.

The patient, H. S—, is a boy, aged 2½ years. His mother says he has always been a healthy child and has always been well nourished. For about a year she has noticed that his left side has been growing faster than the right side. She brought him to the hospital because he walked lame owing to the difference in length of the two legs. There is no mental deficiency, and the child appears to be well nourished and in good health.

The whole of his left side is larger than the right. The left leg is one inch longer than the right, and a secondary curve has formed in the tibia, which is probably compensatory. The left upper extremity is half an inch longer than the right. The girth at the umbilicus is one inch greater on the left side. The girth of the chest at the nipple line is one inch bigger. The girth of the skull at Read's base-line is half an inch more. The girth of the limbs on the left side is considerably bigger. The left side of the face is larger every way than the right. The left half of the tongue is longer and broader than the right.

An X-ray photograph of the limbs shows that the bones are larger on the left side.

The bend in the left tibia suggests rickets, but there are no other traces of that disease, and it seems more probable that the curve is compensatory.

There is no muscle-wasting or evidence of nerve-lesions, and to a physical examination all the organs appear to be healthy. (*Exhibited January the 19th*, 1906.)

Discussion.

Dr. A. C. E. Gray asked how it was decided that the case was one of hemihypertrophy and not hemiatrophy, and whether there were any suggestions as to the causation.

Mr. N. Bishop Harman said he noticed that the left or larger side was much less active and strong than the other.

Both hands could be used with freedom, but the left was flabby and seemed deficient in power.

Dr. George Carpenter, in reply, said he must leave the question of atrophy or hypertrophy to be decided by the questioner, as he did not feel equal to the occasion. He was told by the nurse that the child was left-handed, but he had not confirmed it. If it were true it would suggest that the left was the normal side. If Mr. Harman's suggestion was correct perhaps the right was the normal side.

46. A CASE OF SUCCESSFUL REMOVAL OF A CONGENITAL SACRAL TUMOUR.

By ARTHUR EDMUNDS, M.S.

The patient, a fairly well-nourished female child, aged 18 months, was brought to the Out-patient Department of the Paddington Green Children's Hospital with a large tumour in the sacral region.

The tumour had been present from birth, but the mother thought that it was slowly but steadily increasing in size. It consisted of two distinct parts, one of which was obviously solid, while the other was definitely cystic. The former was situated slightly to the right of the middle line, overlapping the right sacro-iliac synchondrosis. The skin over it was slightly more hairy than the rest of the body, but otherwise it was quite normal. The tumour itself was about the size of an orange, firmly fixed to the skin, while a fair amount of mobility could be obtained on the deeper tissues.

Below and to the left of this, overhanging the anus, was an ovoid swelling, darkish in colour, and presenting all the characteristics of a thin-walled cyst, not very tense, and covered with thinned skin.

A rectal examination was made, and the hollow of the sacrum could be felt free from the tumour, so that it seemed that it would be feasible to remove the whole tumour without any great difficulty.

Accordingly, a curved incision was made along the upper and right border of the swelling and the whole tumour, including the cystic portion, dissected back from the sacrum.

Little difficulty was experienced in doing this until the coccygeal region was reached, and then it was found that the deep attachments became much more intimate. The coccyx was, therefore, cut across at the junction of the second and third pieces, when a fibrous cord came into view, extending up into the hollow of the sacrum. This was defined, and it was soon possible to make out the limit of the tumour and to cut the pedicle across. No important vessels needed ligature. The sides of the cystic portion could now be freed and the cystic portion peeled off the deep surface of the skin without any difficulty. The operation was completed by a transverse division of the skin at the line of junction of the two portions of the tumour.

This rather roundabout method was adopted as the skin over the cyst was thin and it seemed advisable not to remove any skin until one could see exactly how much would be required to close the wound accurately and without tension. The skin over the tumour was quite free from the tumour, so that its vitality was not interfered with, in spite of its thinness.

The specimen exhibited shows a section of the whole tumour and the terminal part of the coccyx. The solid portion is seen to consist of fat pervaded by coarse strands of fibrous tissue. In one place a small bone was found, with cartilage-covered ends. The section shows a small nodule of cartilage. Running through the centre of the tumour there is a branched tube containing the small, fatty, epithelial balls, which are characteristic of dermoid tumours. The lining epithelium is of much interest inasmuch as parts of the tube are lined with typical stratified epithelium, while others are lined with a columnar epithelium with tubular glands, the whole closely resembling the lining of the large intestine. Beneath the lining epithelium there are sparsely-scattered glands.

No definite nervous tissues could be made out, but there are some masses of reticulated tissue which may, perhaps, be an attempt at the formation of neuroglia.

The cyst contained semi-purulent material, and at the junction of the two portions there are a number of smaller cysts.

The child, after the removal of the tumour, did well, the wound was drained for forty-eight hours, and, in spite of the proximity to the anus, healed normally. The child is now well and is, indeed, in better health than before the operation. (*Exhibited January the* 19*th*, 1906.)

Discussion.

Mr. Thomson Walker said the case was a very interesting one, and he would have liked to have heard Mr. Edmunds' idea as to the pathology of it. One found in that region, under the name sacro-coccygeal tumours, a very large number of tumours of different tissues, some mixed, and some apparently pure. The present seemed to be one of the mixed sort. Their origin had been ascribed to the post-anal gut, and to the coccygeal gland, amongst other things. He did not think the mixed form could be properly ascribed to either of those structures. They formed a series of tumours which in their simplest form were lipomata, in their true complex forms appeared as mixed tumours, and in their higher forms showed the formation of fully-developed organs and tissues. These formed a series of tumours which were to be ascribed to fœtal inclusion, teratomata in short. But in that region one also found pure dermoid tumours, and if an explanation of the origin of these were required one could find it by making sections of the skin in that region in infants. In nearly all infants the skin dipped down at the tip of the coccyx, causing post-anal dimples. Sometimes several of these dimples might be found. The tumours arising from these did not necessarily grow during childhood. Recently he had removed one from a lady, aged 54 years. It was a small pure dermoid tumour, and he removed with it the tiny coccygeal gland which was not a part of the tumour, but came away with it. Further there were a few sacro-coccygeal tumours which might be ascribed to the coccygeal gland. Such tumours were rare, but he had seen a few of them. In structure they resembled growths arising from the suprarenal capsule, the inter-carotic body and some other ductless glands. He asked what Mr. Edmunds' idea was as to the origin of the tumour which he had exhibited?

Mr. Arthur Edmunds, in reply, said he regarded the tumour as a true teratoma, or fœtal inclusion. Most of the mixed tumours had not the intimate relationship with the skeleton which the present one had. The fibrous cord which went on to the ventral surface of the sacrum indicated that here there was an attempt at bifurcation of the axial skeleton, and therefore it was probably not one of the mixed tumours derived from structures, such as the coccygeal gland, post-anal gut, etc. It probably represented what would have become an attached twin, similar to the famous Siamese twins, had the conditions under which it developed been more favourable.

47. AN INFANT WITH CONGENITAL ABSENCE OF THE SKIN.

By J. G. EMANUEL, M.D.

Dr. J. G. Emanuel showed an infant with congenital absence of the skin affecting the hands, legs, and feet. In addition, over the temples, the bridge of the nose, and the middle of the lumbar region, there were irregular-shaped areas in which a similar congenital deficiency in the formation of the epidermis existed. The parts affected were smooth, free from hair, sharply defined from the neighbouring healthy skin, and transparent so that the delicate underlying blood-vessels were clearly visible. The infant was born dead at full term. There was no history of syphilis, no signs of any skin eruption, and no other congenital malformations were present. Microscopic examination of sections showed that the defect consisted of a complete arrest of development of all layers of the epidermis including its associated glands (sebaceous and sudoriparous) and hairs. The cutis vera was unaffected except, of course, in so far as it lacked hair-follicles, sweat and sebaceous glands.

(*Exhibited January the* 19*th*, 1906.)

Discussion.

Dr. Colcott Fox said he did not think he could throw any light on the nature of this particular case. But there was one possibility which should be kept in mind when a child was born with excoriated patches. There were two bullous affections which might be met with absolutely congenital, namely, bullous syphilide, and the congenital condition of the skin known as epidermolysis. In the latter affection the children had bullæ form from the slightest traumatism, leaving excoriated patches such as in ordinary pemphigus. It was conceivable that that process might have been going on in the fœtus for some time before birth. He did not suggest that syphilis was the cause of the present case.

Mr. George Pernet regarded the case as unique. Dr. Emanuel had kindly allowed him to have some sections of parts excised, and from them Mr. Pernet concluded the case was one of arrested development of the epidermis. In two of the sections there were no structures developed from epidermis in the corium, but at one point in two other sections there was a hair follicle cut across in the corium and he could only explain it by it having possibly been in the third dimension, that is, probably the section included part of a hair which came from a normal piece of skin adjacent to the portion excised by Dr. Emanuel. In the sections a very narrow flat limiting membrane was made out where the epidermis was wanting. There was no trace of papillary formation. In the case recorded by Hans von Hebra* the epidermis was present and it was made up of three layers, so that case was not on all fours with the present one. In connection with Hebra's case Unna† mentioned that as there were these three layers of epidermis instead of being arrested development, the defect was really a healed surface. Mr. Pernet did not consider it was either a bullous syphilide or an instance of epidermolysis bullosa.

48. A CASE OF CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By GEORGE CARPENTER, M.D.

Alice B—, was admitted into the North-Eastern Hospital for Children on January the 1st, 1906. Her age was 4 weeks, and she was thin but not markedly wasted. She was born fat, and appeared healthy, but ever since the first two days after birth she had been "sick after each feed"; she was breast-fed. A week prior to her admission she "vomited immediately after nursing." Her bowels had been confined, and when opened the motions were like "green water," and for three days before she was brought into hospital she had had no action. Another child died of wasting at three months old, fourteen years before.

On admission she weighed 5 lbs. 12 oz., the pulse rate was 96 to the minute, the respiration rate 32, and the temperature 97° F. Soon afterwards she vomited a large quantity of undigested milk, much more than the previous feed would account for, and the bowels acted freely. A lump was found in the abdomen, about the size of the terminal phalanx of the little finger, hard but not of stony hardness, and with a ridge round it acorn-like. It lay above and to the right of the umbilicus, deep down in the abdominal cavity, very deep, right on the spine, and could be grasped with difficulty between the finger and thumb. When grasped it was obvious enough, but it would have been quite easy to miss it, and it required the exercise of great care and patient manipulation for its detection. The stomach walls also felt thick. The child was first examined without an anæsthetic, but subsequently chloroform was administered without, however, facilitating the examination. There was visible peristalsis of the stomach, and this could be also produced by flicking the abdominal walls.

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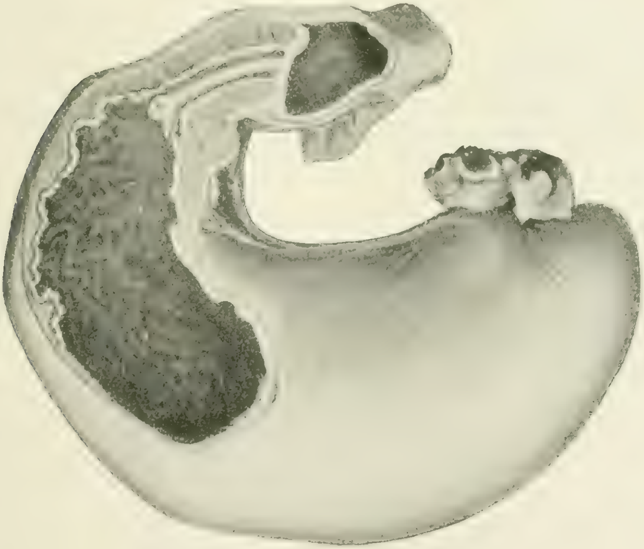
* Hans von Hebra, 'Mitt. d. Wien. Embryol. Inst.,' 1882, vol. ii, p. 2 (cited).

† Unna, 'Histopathology of the Diseases of the Skin,' 1896, p. 1181.

Fig. 8.



Apart from the condition of the pylorus, nothing abnormal was found. She was ordered a whey and cream mixture, and the stomach to be washed out twice daily. On January the 2nd she was sick three times, and the bowels were opened once. She was taking a mixture of tincture of opium, a third of a minim thrice daily for a dose. On the 3rd she vomited eight times, and the bowels were not opened. She had lost half a pound in weight since admission. On the 4th she vomited twice, directly after being fed. The bowels were opened three times. She had gained a quarter of a pound in weight. Olive oil was given in place of the cream, sufficient to make a mixture containing 3 per cent. fat. On the 5th she lost one ounce, on the 6th two ounces, and on the 7th the weight remained stationary at five and a quarter pounds. The olive oil in the milk mixture was thought not to be a success on account of the sickness which had increased to nine times on the 5th, with one exception immediately after feeding, and as on the 6th the vomiting had not improved much, it was omitted and cream substituted. On the 7th she vomited eight times, mostly after feeding, and twice the vomit was a "brown fluid." During this period stomach washings were continued, and saline injections were given by the bowel. Albumin-water was substituted on this date for the whey mixture. The bowels acted twice daily on the 5th, 6th, and 7th. She retained the albumin-water much better than the whey and cream, and only vomited once or twice and in small quantities. Although there had been no further loss of weight the infant's appearance was not satisfactory, and it was weaker than on its admission. Dr. Carpenter, therefore, decided to have the child operated upon, the operation being performed by Mr. Ewen Stabb. Ether was administered by the open method, with the occasional addition of a few drops of chloroform. The pylorus was found to be very much thickened, hard, and about the size of an almond-nut. It was decided to perform a posterior gastro-jejunostomy, the pylorus being thought to be too thick and hard for pyloroplasty. The stomach walls were considered to be thickened. Two ounces of whey were injected into the small intestine, which was contracted and looked empty, the stomach, on the contrary, required the withdrawal of some of its contents to facilitate manipulations. The abdominal cavity was well flushed out with normal saline, a quantity being left in the abdomen. The child vomited several times and the pulse became very weak; on getting her back to her cot it was scarcely perceptible, and a mixture of normal saline, adrenalin, and brandy were injected slowly *per rectum.*

This was not retained, so 8 ounces of normal saline were injected into the subcutaneous tissue. This had a good effect on the pulse, which became stronger and able to be counted. But she continued to vomit frequently a dark brown material. After one of these attacks the pulse suddenly became worse and could be felt only occasionally; she did not rally, and died five or six hours after the operation.

Post mortem.—The stomach was not dilated. Water poured into the stomach through the œsophagus, first distended the viscus, which held $1\frac{1}{4}$ ounces, and then passed out slowly through the pylorus. When the funnel was raised to the height of 2 feet the fluid escaped by drops only. While the passage of fluid was slow the obstruction did not appear very great, five small knitting-needles were passed through the pylorus easily, as also a No. 2 catheter, and the diameter of its lumen was 2 mm. The indurated pylorus measured $\frac{3}{4}$ths inch by $\frac{5}{8}$ths inch, and the stomach walls were thickened. The operation sutures held well, and there was no leakage. There were no other abnormalities.

(*Exhibited January the* 19*th,* 1906.)

49. DRAWINGS OF A CASE OF VACCINAL CONJUNCTIVITIS, WITH NOTES.

By N. BISHOP HARMAN, B.C.

(*Exhibited January the* 19*th*, 1906.)

Discussion.

Dr. Colcott Fox asked whether he understood aright that the lesions were typical vaccine vesico-pustules, because the sketch handed round immediately suggested ordinary streptococcic impetigo, which so frequently followed vaccination, and he gathered that no scars resulted. It was not quite clear to him at what stage of vaccination the condition began to arise.

Mr. N. Bishop Harman, in reply, said he ought to have mentioned that the sores came up at the same time as the vaccination scar. They were first blebs, which burst, and then they became very impetiginous, but they were very deep and ulcerated into the true skin and left deep scars, exactly like a vaccination mark. The boy had lost an eye, and his face was horribly scarred. A bacteriological examination was made, and it was found to be a mixture of cocci. There could be no doubt it was either diphtherial or vaccinal, because there was necrosis of the whole skin, leaving a greyish mass.

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50. MILK CYST IN THE BREAST OF A MALE CHILD, AGED EIGHTEEN MONTHS.

By GEORGE CARPENTER, M.D.

Edward T—, aged 18 months, a well-nourished and healthy child, was brought for advice in regard to a lump in his right breast which had been noticed since birth and which recently had decreased somewhat in size. The tumour was the diameter of a two-shilling-piece, freely movable, soft, and elastic. The overlying skin was natural and non-adherent. On exploratory puncture a milky fluid was withdrawn, which gave the microscopical and chemical tests for milk. (*Exhibited January the* 19*th*, 1906.)

51. HYPERTROPHY AND DILATATION OF THE HEART IN A CHILD WITHOUT VALVULAR DISEASE OR GENERAL ADHESION OF THE PERICARDIUM.*

By GEORGE CARPENTER, M.D.,
AND
THEODORE FISHER, M.D.

Occasionally cases of death from cardiac disease are seen in the post-mortem room not only in adults but also in children in which the ordinary well-recognised morbid affections of the heart are absent. Although such cases are rare, at least in children, that they do not attract attention more often is no doubt due to the fact that the idea that even slight degrees of mitral regurgitation are sufficient to account for death is deeply sunken in the medical mind. An apical systolic murmur has been heard during life, and after death deformity of the mitral cusps is looked for and not, perhaps, uncommonly found without sufficient reason. One often hears of the expression "thickened edge of mitral" as if the thickened edge were sufficient to account for death. The so-called thickened edge is frequently merely the normal thickening which is situated where interlacing of the fibres of the chordæ tendineæ takes place at their insertion into the mitral cusps. This is made perfectly clear on holding up the large mitral cusp against the light, when the course of the fibres of the chordæ tendineæ between the layers of endocardium forming the cusps can be readily seen. Although, however, we have frequently seen this normal band of thickening considered to be evidence of disease, it is needless to say that abnormal thickening may be present in the same situation. When thickening of the mitral valve occurs it affects the fibres of the chordæ tendineæ from their origin at the apices of the musculi papillares to their termination at the mitral ring. As might be expected, this thickening of the fibres of the chordæ tendineæ is most evident where the normal interlacing takes place—that is, at their insertion into the cusps of the mitral valve. Such a band of thickening cannot, however, interfere with the action of the valve. Should regurgitation be present it is generally mainly due to shortening of the chordæ tendineæ, which shortening prevents efficient closure of the cusps. Slight degrees of mitral regurgitation can, however, hardly account for death, and certainly cannot explain considerable general dilatation of the heart.

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* See 'Lancet,' May the 19th, 1906, p. 1385.

In the case recorded below slight thickening of the mitral cusps of the character which we have indicated was present, but was not sufficient in degree to have interfered in any way with the action of the valve. Mitral regurgitation was present, but in this case it was secondary to dilatation of the orifice, not to shortening of the chordæ tendineæ. The following are brief notes :

A boy, aged 8 years, was sent to the North-Eastern Hospital for Children on September the 10th, 1905, by Dr. John Spurway, of Brondesbury, to be admitted under the care of one of us (G. C.). One year previously the boy had suffered from slight "heart trouble," and four months before admission there had been swelling of the elbows, knees, and hands. On admission the cardiac impulse was in the left fifth interspace outside the nipple line. A systolic murmur was present at the apex, and a reduplicated second sound over the pulmonary area. On November the 6th the liver was noted as being 2 inches below the costal margin, the cardiac dulness extended also over 1 inch to the right of the sternum. In addition to the systolic murmur at the apex a to-and-fro sound was heard at the base which Dr. Carpenter considered might be either endocardial or pericardial. The pulse was 140. Some œdema of the legs was present. On the 30th the basic murmur was still present and the pulse was still 140. The œdema of the legs was less. On December the 11th it is interesting to note that although the boy was in other ways worse, the œdema of the legs had disappeared. He died on January the 2nd, 1906.

At the necropsy the body was found to be wasted. There was no trace of œdema of the legs. The heart was much dilated. It extended from the junction of the cartilages and ribs on the right side to 1 inch outside the nipple line on the left; it weighed $10\frac{1}{2}$ ounces. The pericardium was perfectly healthy, except that a few small petechiæ were present over the back of the auricles. Nothing could be found to account for the basic murmur. The pulmonary and aortic valves were both competent to the water-test and the cusps were free from thickening. The tricuspid orifice was much dilated; it admitted four fingers, but the cusps were healthy. The mitral orifice was also dilated; it admitted three fingers. The chordæ tendineæ were slightly thickened but not shortened. There was no deformity of either the large or the small flap. "Water-marking" of the interior of the left auricle showed that there had been regurgitation through the mitral orifice during life. The cardiac muscle was pale, but to the naked eye there was nothing definitely abnormal. The liver extended 3 inches below the costal margin; on section it presented the characteristic nutmeg appearance. There was nothing noteworthy about any of the other organs except the lungs, which were dotted over with small circular petechiæ. Microscopical examination of the heart muscle showed a few fibres in which fatty degeneration was considerable and many others in which small granules were scattered throughout the fibres, but the amount of fatty degeneration was not greater than is frequently seen in death from almost any cause. There was little or no increase of interstitial tissue. The microscopical examination threw no light upon the nature of the hypertrophy and dilatation.

As has already been mentioned, the main morbid condition present was dilatation, associated with hypertrophy of the heart, and the question of the causation of this dilatation and hypertrophy presents itself for consideration, as it does in all cases in which valvular disease and general adhesion of the pericardium are absent. In adults there are several causes of such a morbid condition of the heart, but in children possibly almost the only cause is rheumatism. Symptoms of cardiac weakness appear to have occurred in this boy before swelling of the joints was noticed, yet the fact that rheumatism definitely had at one time been present strongly suggests that there had been also an earlier attack which had fallen mainly upon the heart.

The idea, however, that rheumatism, apart from valvular disease and general adhesion of the pericardium, can cause enlargement of the heart may appear fanciful. Let us consider for a moment, however, the enlargement which occurs in association with pericarditis due to rheumatism. In such cases not only dilatation but hypertrophy of the heart may be remarkably rapid. We have seen pericarditis develop in a girl, aged 11 years, while under treatment for chorea, and cause death within a fortnight, at the end of which time the heart was more than double the normal weight for a child of that age, and such an event, which was noticed many years ago by Dr. J. F. Goodhart, is by no means uncommon. No other disease but rheumatism produces such a rapid increase in weight of the heart. It may be said that such rapid increase of weight must be due to imbibition of fluid. That may be so, but the heart does not return to a more normal size when the inflammation of the pericardium has subsided. On the contrary, the hypertrophy is generally progressive and the enlargement may become so great that even a child 12 years of age may possess a heart weighing 30 ounces. Barnard, Sequeira, and others, have given a simple explanation for the dilatation of the heart in pericarditis. They consider that the pericarditis causes softening of the fibrous pericardium, which yields and allows dilatation of the heart. Hypertrophy follows this dilatation. If, however, softening of the fibrous pericardium were the explanation of dilatation of the heart associated with pericarditis, the dilatation should occur not only in rheumatic pericarditis but in other forms of pericarditis. But in forms of pericarditis equally, if not more virulent, such as the suppurative pericarditis of pneumonia, or of pyæmia, little or no dilatation of the heart takes place. This absence of dilatation in suppurative pericarditis one of us (T. F.) has long pointed out in the post-mortem room and mentioned it in reply to Dr. J. H. Sequeira at a meeting of the Society for the Study of Disease in Children in October, 1900. It is interesting to note, however, that Dr. G. F. Still has since commented upon the same fact.*

Since the difference in degree of the dilatation of the heart which occurs in association with rheumatic pericarditis and in suppurative pericarditis is very great, it seems clear that the dilatation rests upon no such simple causation as yielding of the fibrous pericardium. It depends, we believe, upon something more subtle, upon a poisoning of the heart which affects it chiefly in this way during the growing period of life. After the age of twenty years rheumatic pericarditis causes little or no dilatation of the heart. Allowing, however, that the enlargement of the heart is due to rheumatic poisoning, it is nevertheless true that such enlargement is rarely seen to follow rheumatism unless pericarditis has been present. But this is not surprising. Pericarditis is, no doubt, an evidence of the most intense form of rheumatic infection of the heart, and to get infection which attacks the cardiac muscle while the pericardium escapes can hardly be expected to be common. It must also be remembered that some general cardiac hypertrophy is not uncommon in mitral stenosis. To explain hypertrophy of the left ventricle on mechanical grounds in mitral stenosis is not easy. It seems far more reasonable to allow that the hypertrophy is the result of some interference with the nutrition of the cardiac wall by the rheumatic poison. But however that may be, cases are undoubtedly to be seen from time to time of enlargement of the heart without valvular disease and without general adhesion of the pericardium in children who have suffered from rheumatism, and it is reasonable, in the absence of evidence of other causation, to attribute that enlargement to the rheumatism.

(*Exhibited January the* 19*th*, 1906.)

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* 'Brit. Med. Journ.,' vol. ii, 1901, p. 606.

52. PHOTOGRAPHS AND NOTES OF A CASE OF ERYTHEMA MULTIFORME.

By PERCY G. LEWIS, M.D.

The photographs shown are taken from A. H—, aged 8 years, a healthy boy of gouty inheritance, fair complexion, who is said to be subject to rashes. He had been a year at his present school, but had not previously had eruptions during term time.

History of present attack.—About ten days previously his parents took him out for the mid-term holiday, and allowed him to feed injudiciously. He was also taken for a motor ride of thirty miles.

On November the 10th he became feverish (temperature 100° F.), and an eruption of small papules occurred over the chest and back. The papules were red and each was surmounted by a tiny vesicle. These did not suppurate, and in three days had disappeared. Then commenced a rash over the legs and arms of a different character. This latter rash had the appearance of vesicles not umbilicated. These suppurated in two or three days, and became surrounded by a ring of inflamed skin. Hæmorrhage took place into many of them, giving a black appearance, and several of them oozed a purulent bloody fluid. The spots varied in size from smaller than a pea to larger than a shilling-piece. On rubbing them firmly with a swab of wet wool, the epidermis peeled off easily, leaving a pale, fleshy growth having the appearance of granulation tissue. The growths were painless and easily cut away with scissors.

General condition.—The boy never felt ill, and his appetite was well maintained all through the attack. For the first ten days he passed large quantities of uric acid crystals in the urine, which was otherwise normal. The fæces were offensive at first, but under calomel and magnesium sulphate rapidly became normal. Diet had no effect on the spots. Various antiseptic ointments were used, but resinol was the only one which had any decided effect. The rash was cured in about six weeks.

Microscopic sections of the masses showed proliferation of the cells of the true skin with immensely hypertrophied papillæ.

Note.—The eruption was evidently of toxæmic origin, as shown by the offensive nature of the stools, and the passage of large quantities of uric acid.

(*Exhibited January the* 19*th*, 1906.)

Discussion.

Dr. George Carpenter asked whether the child was taking any drugs—for instance, bromide of potassium. The picture suggested to him a bromide rash. He believed Dr. Lewis said the sores fungated, and that was very suggestive of a bromide eruption.

Mr. George Pernet said the question of drugs as a causation had also occurred to him, but his final conclusion was that it was erythema multiforme resulting from food-poisoning.

Dr. Colcott Fox thought Dr. Carpenter's suggestion was very pertinent. There were certain vesicular and bullous eruptions which exceptionally vegetated, such as a variety of pemphigus and impetigo contagiosa, but the common one was due to bromide of potassium. Therefore he would be interested to hear whether the child was taking any bromide.

Dr. Percy Lewis (Folkestone), in reply, said the child was eight years of age, had been perfectly well at school, there had been no previous illness, and it was not taking any drugs whatever. Until the mother fed him enormously at the hotel and took him for a motor ride, and then fed him again, he was in sound health.

53. CASES OF MENINGOCELE OCCIPITALIS, MENINGOCELE LUMBOSACRALIS, AND THYREOGLOSSAL APPENDAGE.

By J. HOWELL EVANS.

My object in bringing these three cases of—(1) meningocele occipitalis; (2) meningocele lumbosacralis; (3) thyreoglossal appendage—forward is that they should serve the text for a few suggestions which I have to make as to the origin of such conditions, and as to nature's art in occluding *congenital protrusion cysts.*

I will first direct your attention to spinal meningoceles, termed spina bifida because of the anatomical condition found in the laminæ of the vertebræ associated with the lesion.

In the case of meningocele lumbosacralis—the most frequent form of spina bifida—the cause is stated to be due to the "non-union of the laminæ of a vertebra or vertebræ." This is a fallacious estimation of "cause and effect." The cyst is not a consequence of the non-union of the laminæ, but its interposition prevents the normal union of these osseous centres.

We are thus able to fix the epoch of origin of meningocele spinalis antecedent to a certain stage in the formation of the neural arch. Now, the ossification in the neural arches commences first in the cervical vertebræ, so that spina bifida cervicalis belongs to a period of embryonic life earlier than spina bifida lumbosacralis (unless associated with an obstruction in the central canal in dorsal regions). Hence it is the protrusion cyst which prevents the union of the laminæ ; furthermore—there is no deficiency of bone in such cases ; "the mass of bone present in a spina bifida is usually as great as when the union of the laminæ is complete."

In the consideration of all congenital lesions two main questions baffle us—(1) How much is due to disease, to the toxicity of definite chemical bodies of primary maternal origin ? These diffusible poisons, transferred to the embryo, stimulate certain epithelial or secreting surfaces with consequent increase of secretion, which may be the cause of the formation of these protrusion cysts ; and (2) How much in the making of man does the human embryo wander among the ghosts of departed types (Drummond) ? "So that every creature that lives climbs up its own genealogical tree before it reaches its mature condition."

Necessarily we find this law of recapitulation somewhat variegated by the undue prominence of characteristics of lower types. I think a due estimation of the value of each of these factors may lead us along the correct track of investigation. I myself most decidedly believe in the transmission from parent to embryo of diffusible toxines which produce many such effects.

I consider that the lining epithelium of the central nervous system—the ependyma—may be stimulated to so great an increase of secretion as to produce a protrusion cyst. I further hold that when the toxines have ceased to act or have been eliminated, the forces of nature—absorption, adhesion, and contractility—come into play. So great is nature's force of contractility that it is the greatest of all healing powers. In the case of a meningocele the contractile tissues occlude the cyst so that there only remains a mass of dense vascularised fibro-fatty tissue with a crenated or puckered appearance.

Your valuable time will not allow me to discourse upon the cranial protrusion cysts, but you will realise from my previous remarks the trend my expressions would take. I must, however, in the brief space of time granted me refer to the thyreoglossal duct.

The ductus thyreolingualis of His, of which the part above the hyoid bone is known as the ductus lingualis, whereas the part below the hyoid bone is called the ductus thyreoideus, is an epithelial structure whence is derived the primitive representative or analogue of the isthmus of the thyreoid gland. This duct should be closed throughout its length, its closure being carried out by two mechanical forces—compression and traction. The compressing force is exercised by the formation of the hyoid bone, the traction is the general traction due to the straightening of the head with the opening out of the cervical sinus, with the growth forward of the tongue and the pulling downwards of the descending thoracic viscera.

As the pharynx and alimentary canal of the child and adult participate in most infectious lesions so the epithelium of the pharynx of the embryo and fœtus becomes affected by the transmitted maternal poisons. The secretion may be in excess of the rate of its absorption, and closure of the duct may be prevented. Various cysts may derive their origin in this manner.

Nature's cure is rupture with subsequent inflammation, but this crude method is extra-uterine. If, however, nature's force of contractility comes into play during intra-uterine life a small puckered lump may represent a protusion cyst of earlier days. Such is the view I am inclined to take of this unusual thyreoglossal appendage and to compare it to the single median wattle of certain birds.

(*Read February the* 16*th*, 1906.)

Discussion.

The Chairman (Mr. A. H. Tubby) said he was sure all present had heard Mr. Howell Evans's paper and seen his photographs with much enjoyment. The author had raised several points which might profitably be debated, and he hoped they would be discussed. He also said he would have liked to have heard more about the toxins which had been referred to, not only in respect of their effects, but what proofs there were of the existence of such poisons.

Dr. Charles W. Chapman asked why, if these cases are the result of poisoning, the cysts should occur at about the same place, namely in the sacral region, rather than higher up.

Mr. Howell Evans, in reply to Dr. Chapman's question, said the view which he had endeavoured to put forward was that the cervical region closed earlier than the lumbosacral region, and accordingly a toxicity which might appear at a very early period would be of such a degree as to destroy the ovum or insufficient to produce a protrusion cyst in the cervical region; but the lumbosacral region, being more slow in its progress, any increased closure of the interspinal exudation might culminate in a protrusion cyst and hence be seen more frequently at this spot. He also hinted in his paper at the possibility that sometimes the reason why a tumour was found in the cervical region was because there was an occlusion somewhere in the dorsal region. Accordingly he suggested there was a date at which one would presently be able to locate these conditions in embryonic life. In reference to the questions Mr. Tubby referred to, he (Mr. Howell Evans) had discussed the question with one of the members of this Society who was a great authority on all toxic conditions, and they were both at present obliged to admit that the connection is not proven. But how was one to account for inflammatory exudations except by analogy? There were many points in favour of and against the views put forward, but to discuss them at the present time would carry him beyond the time-limits of the Society.

54. TWO CASES OF JAUNDICE IN CHILDREN.

By F. J. POYNTON, M.D.

Persistent jaundice in infancy is such a dangerous symptom that these two cases which have come to my notice in the last two years are, I think, of practical interest. They are cases with which I have had little acquaintance, and about which I am anxious to learn.

You will pardon the lack of detail in their clinical descriptions when I tell you that both occurred in private practice and that the complete notes were apparently destroyed, in the first case by someone in the house, who disliked the idea of any illness having occurred in their family.

This first case, which I saw in consultation with Dr. Wightwick, was a female infant, aged 3 months. She was then distinctly jaundiced, and had been so since birth. The conjunctivæ, mucous membranes, and skin were yellow; the motions, as a rule, were undigested and white, and the urine very dark. In fact, there was well-marked obstructive jaundice, which had lasted from birth. The liver was large but not tender or unusually hard; the spleen was not felt, there were no hæmorrhages, and the umbilicus was healthy. The temperature was generally subnormal, but sometimes a little above the line.

The family history threw no light upon the cause; the parents were well-to-do, there was not the least evidence of any specific disease, and there was one other child—a boy—quite healthy. The child had not been breast fed.

The jaundice had, on the whole, diminished a little since birth, although it was at three months very definite. The most interesting feature in the case was the occasional appearance of a little bile in the stools, with a concomitant improvement in the colour of the skin.

The great practical difficulty was feeding the child. Fats and proteids were passed in the motions undigested, and, although there was no serious vomiting or diarrhœa, the child did not gain, and suffered from flatulence. The prognosis, too, was a difficult one. We had mainly three facts to guide us in making a forecast :

(1) That a jaundice of considerable intensity and dating from birth had persisted for three months and was still serious.

(2) That no cause could be ascertained for the jaundice.

(3) That bile occasionally appeared in the stools, and that on the whole the jaundice had slightly diminished.

I came to the conclusion that there was probably no obliteration of the bile-ducts, and attributed the condition to a viscid bile, together with possibly unusually small biliary channels.

That, as Dr. Still pointed out in 1901, the bile may be viscid in infancy, I assured myself eight years ago, at a post mortem I had made on a fatal case of icterus neonatorum. In that case the bile was like tar, and there was no other cause that I could find for the jaundice. This hypothesis had the advantage of holding out some hope that the child might recover, and encouraged treatment.

The diet that we found very useful was asses' milk and Benger's food, or cow's milk and Benger's food, alternating with equal parts of chicken tea and whey. Brandy was given in 5 to 10 minim doses to stimulate the liver, and bicarbonate of sodium to liquefy the bile.

By these means the child began to gain weight and the dangers of undigested fat were in great part obviated. From time to time there were slight checks and the weight then did not advance, but the general tendency was forward. Gradually the jaundice became less distinct, although the stools remained very pale, and a change was made to pure asses' milk, which, on account of its digestibility and slight percentage of fat, is an admirable food for such a case.

Before the child was twelve months old the jaundice had disappeared, and now I am informed that she is a particularly healthy little girl.

I would also add that citrated cows' milk is a useful food for these cases when the jaundice shows signs of distinct improvement.

The second case I saw with Dr. Bott last year. This child, a boy, aged 4 weeks, was the first child. The parents were both healthy and well-to-do, and no sign of a congenital taint could be discovered. No cause at all was forthcoming for the jaundice, which had appeared upon the third day and had persisted.

The child was markedly jaundiced—skin, conjunctivæ, mucous membranes, and urine all proved that the condition was obstructive, and the stools were large, undigested, greasy, and white.

The liver was large and not tender, the spleen was not felt, and there were no hæmorrhages. The umbilicus showed still a raw surface, from which a little clear discharge exuded. This, though not satisfactory, had been recognised and treated, and had never shown any sign of infection. The appetite was good, but the child had only gained 3 ounces since birth, the motions being large and quite undigested.

One feature, noticed by Dr. Bott, the nurse, and parents independently, was the occasional appearance of a little bile in the stools, with a concomitant lessening of the jaundice.

Encouraged by the first case, I made the same suggestion and we carried out the same line of treatment.

Two months later I saw the child again, and, though still jaundiced, the motions were mustard-coloured and the liver distinctly smaller. In this case modified milk answered admirably.

I have heard recently that the jaundice has now disappeared and the child vastly improved, although there is still some deficiency in bile.

I should be most interested to hear the experience of others with these cases, the views they have taken of them, and their methods of treatment.

(*Read February the* 16*th*, 1906.)

Discussion.

Dr. Theodore Fisher said he had heard of similar cases clinically, and had seen such in the post-mortem room. In one case a medical man was very much concerned about jaundice in his child, thinking there might be congenital obliteration of the bile-ducts; but in three or four months the jaundice disappeared. He saw three cases in the post-mortem room, but he did not pay as much attention to them as he might have done. He only cut sections of one of the livers, and that showed early biliary cirrhosis. There having been some inflammation about the umbilical cord, it occurred to him that it might have originated in some septic intoxication in connection with that inflammation. He thought it possible there might be such a thing as biliary cirrhosis due to some form of septicæmia occurring in very early infancy, leading to the production of jaundice. The inflammation of the liver might become quiescent and the jaundice disappear.

Mr. Milner Burgess (Harlesden) said he found the condition not very uncommon in babies, especially those which were hand-nursed. It lasted sometimes for many months, but gradually passed off. The stools of such cases were white.

Dr. F. J. Poynton, in reply, said he was very interested indeed in Mr. Burgess' remark that the condition was common, and especially that it lasted so long. He (Dr. Poynton) very rarely indeed saw such cases. The fact that the liver enlargement so steadily disappeared and that the child was so healthy, made it unlikely that there was anything like actual cirrhosis; but a condition might have been present which might possibly have ended in a cirrhosis, he thought was feasible.

55. A CASE OF SYMMETRICAL ALOPECIA AREATA NEUROTICA ACCOMPANIED BY A BULLOUS ERUPTION ON THE FACE.

By W. GIFFORD NASH.

A GIRL, aged 14 years, was seen on November the 13th, 1902, suffering from an eruption on the face and loss of hair.

The history was that on November the 7th, a lock of hair along the course of the right supra-orbital nerve fell out, and on November the 11th, blister-like spots began to appear on the face. This alarmed the child's relatives, as two years previously she had suffered from similar spots on her face and hands. These had occurred over a period of some months, and had left brown stains. Six years previously she had ringworm of the scalp at the same time as her brothers and sisters. The medical man who attended the child during the first attack, informed me that "the eruption resembled pemphigus. It would appear in a few hours as vesicles, then in a few hours these scabbed over, fell off, and left a brownish stain." In the attack which I watched, the eruption followed much the same course. First a slight burning feeling occurred on the skin of the face, and at this spot a bleb quickly formed and burst, so that in a few hours nothing but a crust or scab was seen, which rapidly fell off, leaving a red mark. The distribution of these bullæ was often symmetrical, within a day or two appearing over the eyes, on the cheeks, or sides of the nose, or in the middle line of the forehead or chin. Altogether up to December the 15th, about twenty blebs formed, the last six appearing in groups of three with a week's interval.

The occurrence of alopecia areata.—As already stated the first patch of hair to fall out was along the course of the right supra-orbital nerve. Ten days later a similar bald patch suddenly appeared on the left side. The symmetrical distribution is well shown in the photographs. For a few hours before a patch of hair fell out a burning sensation was felt over the area affected. This was generally perceived at night, and when the hair was brushed next morning the lock of hair fell out. The skin of the affected patches looked smooth, white, and shiny, and was quite free from scurf. A few club-shaped stumps were found around the edges of the patches, and some of the long hairs adjacent were easily detached. Sensation in the bald areas was very defective.

Dr. Horace Savory examined the short hairs and stained them for tricho-phyton, but no spores were found. The stumps showed atrophied roots and were dilated in places with pigment collected into masses. In fact, the stumps exactly answered the description of those found in alopecia areata given by Alder Smith.

Further history of the attack.—In January, 1903, the patient had measles, but this had no apparent effect on the disease. During the following two or three months, there were fresh patches of alopecia, the hair falling out over the occipital nerve and also a second time over the supra-orbitals. Symmetrical lesions also appeared on the face. The attacks appeared to be caused by the excitement of a children's party or the worry of lessons. The same effect was noticed during the first attack, when it was found necessary to cut off the excitement of juvenile parties.

Diagnosis.—Tinea having been excluded, the only disease of the hair under which this could be classed was alopecia areata. The sudden appearance of the baldness over the course of the cranial nerves and its symmetrical distribution suggested a trophic influence. The child appeared otherwise in perfect health, and had not suffered from neuralgia or headache. The trouble came on in term-time and improved during the Christmas and Easter holidays. I am inclined to think that the cause was entirely central and produced by anxiety and excitement. Up to the present time there has been no return. This affection is classed by Crocker under alopecia areata neurotica varietas localis.

It is very rare, and Crocker says some cases have been preceded by severe and persistent neuralgia or by injury. The bullous eruption appears to have been of the nature of pemphigus and would be called "Pemphigus hystericus," which Crocker says is a variety of Pemphigus neuroticus. It is a rare bullous tropho-neurosis which occurs in women, mostly young, who are pronounced hysterics.

Except for these two attacks of pemphigus and alopecia the patient has never shown any other sign of hysteria or neurosis.

(*Read February the 16th,* 1906.)

Discussion.

Mr. George Pernet considered the case a very peculiar one, especially as the patient was a female, and the condition was said to be worse during term-time and better during the holidays; it had occurred to him that it might be a case of malingering. He had never seen a case of alopecia areata of neurotic origin, so-called, with similar areas of denudation. A point in favour of alopecia areata were clubbed stumps with atrophied root ends. He would be glad to hear if there was evidence of anything having been done to remove the hairs artificially. Physiologists asserted there were no such things as trophic nerves, therefore it would seem the term "tropho-neurosis" must be dropped. It was known that alopecia areata occurred in children who had had ringworm. In women with dermatitis artefacta, who had hysteria, he had frequently found marked ovarian hyperæsthesia, hemianæsthesia of the tongue, or limitation of the field of vision, and so forth. Had these points been investigated in Mr. Nash's case? The case was so unusual that Mr. Pernet was of opinion that malingering was very probably the explanation.

Dr. Porter Parkinson said that some time ago, before the skin department was differentiated from others at his hospital, he saw many skin cases and was very much interested in a case of alopecia areata in a patient who had also developed leucodermia. The latter condition was supposed to be connected with a nervous condition, and was another instance of two different skin affections of trophic causation occurring simultaneously.

Mr. Gifford Nash (Bedford), in reply, said he did not propose to fight out the question concerning tropho-neurosis with Mr. Pernet; he would leave that to Dr. Crocker, who used the term. He showed the photographs to Mr. Jonathan Hutchinson, who suggested that the lesions were consequent upon the ringworm, which the patient had had four years previously, and he also said that the child might be malingering. He (Mr. Nash) felt sure that the child was not malingering, and that the lesions were not self-produced. If hairs were pulled out there would be some signs of hæmorrhage left behind, and an application would not leave the skin white and shiny and absolutely devoid of any hair. Again, if it were self-caused why should a child who knew no anatomy pick out the course of the supraorbital nerve, in which region also a bullous eruption appeared? Why, also, should the occipital nerves be selected? He was certain the condition was due to some disease, and was not caused by any external application.

56. NOTE ON A CASE OF SPLENOMEGALIC BILIARY CIRRHOSIS IN A BOY, AGED SIX YEARS.

By EDWARD CECIL WILLIAMS, M.B.

My patient, a boy, aged 6 years, was admitted into the Royal Hospital for Sick Children, Bristol, on October the 6th, 1905, for enlarged liver and jaundice. His mother died of consumption, his brothers and sister are both alive and healthy. There is a doubtful history of paternal syphilis. The child comes from a village in the Cotswolds. From birth he has been pale and unhealthy. Two and a half years ago he had jaundice, and was then said to be suffering from enlarged liver; since then child has been yellow; the urine has never been dark brown. Appetite good, no sickness or diarrhœa, bowels regular, motions inclined to be white; never had any pain except occasionally in left side; has suffered from shortness of breath, can now run a short distance; legs never swell, has had blue patches the size of a shilling on his legs and abdomen, which are very tender, but are not raised above the skin level. No epistaxis. On admission, child was pale, conjunctivæ yellow, skin of a lemon tint; there were enlarged veins over the chest and abdomen. Heart: apex beat in fifth interspace in nipple line, hæmic murmur at base. Lungs: breath sounds normal. Height: 3 ft. 6 in. Weight: 34 lb. 12 oz.

Abdomen very distended, girth 26 in., enlarged surface veins. Liver: much enlarged, dulness extending from sixth rib to the level of the umbilicus, where its free edge can be felt; left lobe can be felt below the ensiform cartilage; spleen also much enlarged, extending from the eighth rib to within a couple of fingers' breadth of the iliac crest in the axillary line; its notch can be felt 2½ to 3 in. from the umbilicus; the organ appears to be dense and smooth. There is no ascites or œdema of legs, but on each leg are a few petechiæ. Urine: acid sp. grav. 1027, no albumin or sugar, trace of bile (Gmelin's test). No enlargement of lymphatic glands, marked clubbing of fingers and toes. Blood: hæmoglobin 62 per cent., red cells 5,400,000, white cells 9545 per c.mm. Differential count:

Polynuclear . . .	38	per cent.
Uninuclear . . .	3·3	,,
Eosinophile . . .	11·6	,,
Lymphocytes . . .	46	,,
Basophile . . .	1	,,

On admission his temperature was practically normal until October the 12th, when there was a rise of temperature over 101° F., with an increase of jaundice. With the fall of temperature the jaundice gradually decreased, at the present time there being only a conjunctival tinge. The motions on admission were pale, now they are yellow. His abdominal girth has diminished by 2½ inches since admission. No hæmorrhage from any mucous surface, except the motions have sometimes contained blood and mucus.

The use of the term "splenomegaly" in connection with cirrhosis of the liver is only justified when the spleen is enormously enlarged, as some splenic enlargement is a common result of hepatic cirrhosis. Cases of biliary cirrhosis associated with splenomegaly have been divided into the following classes:

(1) Splenomegalic biliary cirrhosis, in which the splenic enlargement is a conspicuous feature.

(2) Hypersplenomegalic biliary cirrhosis, in which the spleen is larger than the liver.

(3) Metasplenomegalic biliary cirrhosis, in which splenic enlargement precedes hepatic.

(4) Hepatomegalic biliary cirrhosis, in which the liver is the prominent feature.

(5) Presplenomegalic biliary cirrhosis, in which hepatic precedes splenic enlargement.

(6) Atrophic biliary cirrhosis, in which the liver is small.

Then there is the juvenile type of biliary cirrhosis, associated with splenomegaly, described by Gilbert and Fournier, of which this case is an example. In this form the clubbing of fingers and toes is a more constant symptom than in adults; the enlargement is not due to bony enlargement. Whether the splenic or hepatic enlargement be primary cannot always be determined. It is probable the enlargement is due to the circulation of some toxin, which may act more selectively on the spleen and liver, and being excreted by the small bile ducts, may cause a descending cholangitis, or it may be due to some poison manufactured in the intestine ascending the smaller bile ducts and so to the spleen along the splenic vein, an ascending cholangitis. Against this view is the fact that the infection would have to travel against the blood-stream. Jaundice is never very deep in these cases, in fact, cases may occur without any jaundice. The child shows none of the signs of congenital syphilis, nor have the symptoms shown themselves amenable to antisyphilitic remedies. So if syphilis plays any part in the causation of the disease it can only be in an indirect manner, and some of these cases may be regarded as parasyphilides. This juvenile type cannot be confounded in its later stages with Banti's disease, as there is nothing distinctive or abnormal in the blood count, and the chronic splenic anæmia of adults does not occur at such an early age. At the present time my patient seems to be improving, whether this improvement will end in cure only time can tell. When these cases tend to go downhill the cirrhosis usually tends to assume the mixed type with ascites, œdema of legs, and often albuminuria. With regard to treatment, salicylate of sodium, 5 grains three times daily, with $\frac{1}{20}$

grain of calomel seem to be useful. Surgical treatment of draining the gall-bladder on the assumption of an ascending cholangitis does not yet seem to be an established practice.

(*Read February the* 16*th*, 1906.)

Discussion.

Dr. Frederick Taylor asked whether Dr. Williams had any reason for believing that the child was of stunted growth or was less fully developed than it should be? Stunted growth was one of the features described by Fournier, and it was marked in the case of the kind which he (Dr. Taylor) published some time ago ('Guy's Hospital Reports,' vols. lii and liv). With reference to Dr. Williams's remark that the jaundice was never deep, he believed his case would contradict that, as it was quite pronounced, and persisted during the whole time the patient was under observation, two or three years. Also, was there any history of any infectious disease which could be supposed to have set up the mischief early in the child's life? As the symptoms began at three years of age there was probably little to help one in that respect. The cases were at present very obscure, and could not be readily explained. Probably there was no information available as to whether these children produced more toxins in their intestines than other people. The recovery from the jaundice was a matter of interest, and made it rather difficult to say how far the cases were all of the same nature, or whether there was any difference between them. In the case which he had himself reported there was very definite cirrhosis after death, the termination taking place some six or seven years from the commencement of the symptoms.

Dr. G. A. Sutherland said he did not think that splenomegalic cirrhosis of a pure form was very common in children, but it was difficult to differentiate them from cases in which there was an increase in size of the liver and the spleen. Three weeks ago he sent a boy, aged 4 years, into hospital, who had great enlargement of the liver and the spleen. Nothing else could be found amiss with the child, and the house physician, who examined him the same afternoon, could not find anything further, and therefore put him upon full diet. At 1 o'clock the following morning the temperature ran up to 104° F., and continued high for a few days. For a couple of days there were no definite signs to be made out, and then there was evidence of lobar pneumonia, and the boy went through a difficult attack. For the first few days after admission the spleen seemed to enlarge still further. After the crisis it was noticed that the spleen began to diminish in size, and in a week both the liver and spleen were much less bulky. The liver extended to 1½ inches below the costal margin, instead of 3 inches as on admission, and the spleen had also diminished. Dr. Frederick Taylor had referred to the possibility of there being some injurious material in the blood. In his own case he thought the pneumococcal poisoning had some effect which led to diminution in the size of the liver and the spleen.

Dr. Cecil Williams (Bristol), in reply, said the height was 3 ft. 4 in., weight 36 lb. 8 oz. He was certainly below the normal. The father's statement was that since the child was taken ill he had practically not grown at all. He did not know whether the boy had had an infectious disease, but he was said to have never been quite well. The boy had been in the presence of an epidemic of measles and of scarlet fever, but without catching either of them, so he may have had scarlet fever and measles.

57. THREE SPECIMENS OF TUBERCULOUS ULCERATION OF THE STOMACH.

By F. LANGMEAD, M.D.

Tuberculous ulceration of the stomach in children is, if we may judge from the published records, a very rare lesion, and few cases have been described by English observers. Dr. Still in the 'Transactions of the Pathological Society of London,' vol. l, gives an account of 5 cases which he had found in 226 autopsies on tuberculous children. He remarks upon the rarity of the condition, and states that he had found records of only 36 or possibly 37 cases. Of these 2 were found by Widerhofer in 418 tuberculous cadavers, 4 by Steiner and Neureutter in 302, 21 by Rilliet and Barthez in 141, 5 by Holt in 119, a doubtful 1 by Sir Thomas Barlow, and 4 others. Dr. Lister in the same volume records 5 examples found among 70 autopsies in tuberculous children. I found the 3 specimens described below among 140 post-mortem examinations of tuberculous children during the last twenty-one months, at the Great Ormond Street Hospital for Sick Children. All 3 were in children under two years of age, 2 being under twelve months, an incidence which is in agreement with that of Dr. Still's cases, 4 of his being under three years, and with Dr. Lister's, none of whose were older than two. The cases described by former writers were also generally infantile. The ulcers were all on the anterior wall or greater curvature, and in this way differed from those described by Dr. Still, which were all on the posterior wall or lesser curvature, but in agreement with the situation of those described by Rilliet and Barthez. It is noteworthy that all were associated with a general blood infection, and the origin is probably to be explained by this fact. In Cases 1 and 3 the situation of the ulcers on the blood vessels strongly favours such an explanation. In Case 1 the nodules are evidently a part of a miliary tuberculosis, and the ulceration is only an added lesion.

CASE 1.—Mary C—, aged 8 months. She was admitted into hospital for whooping-cough, which had started at four months of age. She had been entirely breast-fed. There were no symptoms referable to the condition of the stomach, vomiting only following the paroxysmal cough. She became thinner, developed signs of pulmonary consolidation, and died two months after admission.

Post mortem.—There was general tuberculosis, the meninges, lungs, spleen, and kidneys being all affected. The whole of the intestine showed small tuberculous ulcers, most crowded in the duodenum and jejunum. The thoracic, mesenteric and lesser omental glands were considerably enlarged and caseous. The stomach was not dilated. On its anterior wall were six shot-like submucous tubercles, each capped by a tiny ulcer, and two more were situated on the greater curvature. Their distribution was that of the arteries, and on holding the specimen up to the light when it was first removed each tubercle could be seen perched upon an arterial twig.

CASE 2.—Stephen B—, aged 1 year and 11 months. He was admitted into hospital for whooping-cough and bronchitis, and died a few weeks later, having developed evidences of consolidation of the lungs and having wasted rapidly. There had been no vomiting.

Post mortem.—It was found that a caseous gland had ruptured into the right bronchus, and had produced acute caseous broncho-pneumonia with softening and cavity formation. There was tuberculosis also of the pleura, pericardium, liver, spleen, and cervical, thoracic and mesenteric glands. Many ulcers were found throughout the small intestine, beginning near the upper end of the ileum. There were none in the large intestine. Perihepatitis and perisplenitis were present, but no general peritonitis. At the middle of the anterior wall of the stomach, about half-way between the cardia and pylorus, was a small tuberculous ulcer stellate in appearance, and surrounded by a slightly raised and rolled edge. The greatest measurement between the outer borders is 9 mm. It could not be detected from the peritoneal surface.

CASE 3.—William T—, aged 7 months. He was admitted into hospital collapsed, with a history of five months' "croupy cough" and wasting. He had never been breast-fed, but for the first four months was fed on cow's milk and barley-water, and afterwards on Nestlé's milk.

Post mortem.—In this case, too, a caseous gland had ruptured into the right bronchus, and acute pulmonary tubercle had resulted, the whole lung being cheesy and softening to form large cavities. There was tuberculosis also of the meninges, pleura, liver, spleen, intestines, and mesenteric glands. In the small intestine, from the jejunum downwards, were many small ulcers, in the large intestine only a few. Near the greater curvature of the stomach, and midway between the cardia and pylorus, were two small ulcers with flat, slightly raised edges, the larger measuring only 4 mm. in its greatest diameter, and being plainly situated on a vessel. The other, which was slightly smaller, was about ½ inch distant. The peritoneal surface was unaffected.

(*Read February the* 16*th*, 1906.)

Discussion.

The Chairman (Mr. A. H. Tubby) asked whether Dr. Langmead had anything to say in regard to perforation of those ulcers.

Dr. F. J. Poynton said he thought the tubercular ulcerations referred to were very rare. When Dr. Still was bringing forward his paper it was a question whether the ulceration was tubercular, and he asked him (Dr. Poynton) to make a section of such an ulcer. He did so, and was interested to find in one of the ulcers great numbers of tubercle bacilli—*i. e.* in the edges. He asked whether there had been any cases in which hæmorrhage was recorded, or whether any particular train of symptoms had been detected in the patients during life.

Dr. Theodore Fisher said he thought the small ulcers which look like enlarged solitary follicles are not very uncommon. The question at one time used to arise at post-mortem examinations whether these ulcers were tubercular or not. It was thought they might be inflamed small lymphoid follicles. The larger ulcers were rare. He had only had one example in which the ulcer approached the size of one's little finger nail.

Dr. Porter Parkinson said that some years ago he showed before the Society a specimen of an ulcer apparently originating in one of the lymphoid follicles, but it was quite different from the present one. There was in that no raised edge, as in the present case, which looked as if there was some new growth around the side. He believed there was no doubt his own case was not tubercular, as that disease could not be found in any part of the body. A section of the ulcer was not examined, but the rest of the body was. In his case the symptoms of the ulcer were hæmorrhage from the stomach, followed by perforation and acute peritonitis. The patient was a child two years and two months old.

Dr. F. Langmead, in reply, said that among recorded cases he could only find one with a history of hæmatemesis, and that was the only symptom which he could trace as being associated with tuberculous ulcer in children. He intended to have the sections cut, and, if it would be to the advantage of the Society, he would show them at a future meeting. When first removed, those ulcers in the first specimen, which simulated enlarged follicles, were quite yellow, exactly like miliary tubercle elsewhere in the body. He had no doubt about their tuberculous nature.

58. TWO CASES OF RENAL CALCULUS IN CHILDREN UNDER TEN.

By W. GIFFORD NASH.

Case 1.—George C—, aged 8 years, was admitted into the Bedford County Hospital on May the 27th, 1905, with a history of abdominal pain and frequency of micturition since an early age. The left kidney could be distinctly felt. The urine was acid and contained pus, but no crystals were found. Radiography gave a negative result.

Operation May the 29th, 1905.—The left kidney was exposed by a lumbar incision, and a stone was felt in its pelvis. The cortex was incised, and a stone weighing 30 grains was removed. Seven small calculi were washed out of the kidney. A tube was inserted and the wound sutured. Urine escaped from the loin for a few days. The child made a good recovery.

Remarks.—The size of the main stone shows that it had been present in the kidney for a considerable time. The presence of the small stones suggests that others may have been present and may give rise to further trouble.

Case 2.—E. C—, a boy, aged 4 years, was admitted into the Bedford County Hospital on October the 11th, 1905, with an indefinite history of renal calculus. For two years he had occasionally passed blood in his urine, and complained of feeling cold in his belly. At times he cried at night and "screwed his legs about." The urine contained a trace of albumin, but no pus or crystals. There were no other symptoms of stone in his kidney, no frequency of micturition, no attacks of vomiting, no swelling, and no renal tenderness.

But there was one definite piece of evidence. Three skiagraphs were taken by Mr. King, our house-surgeon, and in each, in the region of the left kidney, was a very distinct shadow.

Exploration of this kidney was decided on, and the kidney was first exposed, and then brought out of the lumbar incision. Some hardness in the renal pelvis was felt, and puncture with a needle showed this to be a stone. A small incision was made in the renal cortex and a grooved director passed into the pelvis; next the wound was enlarged with a blunt forceps and the tip of the index finger. The stone, which was felt lying in the renal pelvis, was seized with a pair of forceps and delivered with some difficulty, owing to its peculiar flat shape. Then followed a continual welling up of venous blood from the kidney. Attempts were made to arrest this by ligature of some vessels, which could be seen bleeding near the hilum of the kidney, by suturing the incision in the cortex and by gauze packing, but without materially lessening the flow of blood. The child was rapidly becoming exhausted from the loss of blood, so I decided to remove the kidney. The vessels and ureter were ligatured and the kidney removed as far from the ligatures as possible. Consequently most of the pelvis of the kidney was left behind. The stone removed consisted of uric acid and weighed 25 grains. It was a flat-shaped stone with a sharp edge. Recovery was retarded by some suppuration, but the ultimate result was good, the child passing about 20 ounces of urine daily when he left the hospital.

Remarks.—I have brought forward these cases partly on account of the rarity of operation for stone in the kidney in children, partly to illustrate the use of X-rays, and to draw attention to one of the dangers encountered in these operations.

The conditions which were present in this case, and which increased the risk of hæmorrhage, may be discussed under four heads: (1) The small size of the kidney; (2) the healthy condition of the kidney; (3) the size, shape, and position of the stone; (4) the position of the incision in the kidney.

(1) The small size of the kidney in a child of 4 makes it difficult to avoid the vessels.

(2) The kidney was healthy, which means that there was no dilatation of the pelvis and calyces and no thinning of the cortex, all of which conditions diminish the vascularity of the organ.

(3) The flat shape of the stone made it difficult to remove through a small opening, and its thin edge made it liable to lacerate the kidney tissue.

(4) The incision into the cortex was a small one on the anterior surface, and was enlarged by tearing.

It would have been safer to have made the incision in a line with the long axis of the organ and on the convex border furthest removed from the hilum, as this would have diminished the risk of tearing a vessel near the hilum.

It may be asked why I did not incise the renal pelvis. To this I answer, that it has always been my custom to make the incision through the cortex, which method is supposed to diminish the risk of a persisting renal fistula.

The second point I wish to refer to is the rarity of operations for stone in the kidney in children.

In Vol. iv of the 'Reports' of this Society, Dr. Parkinson and Mr. Drew related a case of renal calculi in a boy, aged 9½ years.

Dr. Holt in his work on 'Diseases of Children,' refers to the frequency of minute granules of uric acid in the kidneys of infants, but only once in over a thousand autopsies found a stone of any size.

Aldebert, quoted by Holt, collected four cases of nephrectomy in children for renal calculi, in which the kidney was healthy, with three recoveries and one death from shock.

Mr. Henry Morris ('Surgical Diseases of the Kidney and Ureter,' vol. ii, p. 58) says that out of ninety-six operations for renal calculi performed by him, not one was under ten. Out of 203 cases of operation for renal calculus collected by him between 1883 and 1893 (182 of which underwent operation) only one case was under ten. In some of Morris's cases the symptoms began before ten years of age.

Mr. George Heaton in the 'Birmingham Medical Review' for 1897, recorded two cases of nephrolithotomy in children aged nine and eleven years.

In the 'Lancet,' vol. i, 1891, p. 1380, I related a case under the care of Mr. Paul Swain, of Plymouth. A boy, aged 7 years, had double calculous pyonephrosis, for which double nephrotomy was performed.

Lastly, the second of my cases exemplifies the value of radiography in the diagnosis of stone, for without the presence of a shadow in the left loin we had no evidence as to which kidney was affected.

In the 'British Medical Journal' of January the 20th, 1906, p. 137, Dr. Mackenzie Davidson has drawn attention to the diagnostic value of X-rays, and published illustrations showing the relative opacity of calculi. In the 'Annuals of Surgery' for December, 1905, p. 864, Dr. Carl Beck, of New York, says that by means of X-rays a definite diagnosis in suspected lithiasis can be made in each and every instance, in other words, a renal calculus must invariably show, provided a calculus is there.

(*Read February the* 16*th*, 1906.)

Discussion.

Mr. Douglas Drew commented upon the almost complete absence of pain in Mr. Nash's second case. He had operated in three cases of renal calculus in children, the one mentioned by Mr. Nash and two others, and the difficulty in all of them was to determine whether there was kidney involvement, and, if so, in which one, because the tenderness on palpation was so slight. He exhibited a radiograph which exemplified one of the points which Mr. Nash referred to. He recorded the case at the Clinical Society last year as calculus anuria in a boy, aged 4 years. He had removed a stone from the bladder, and cystitis, or what was regarded as such, persisted for a time. Upon examination of the rectum after some weeks he found there was evidence of stone in the left ureter, although the skiagram which was taken was not sufficiently good to reveal the calculus. The stones were removed, but the case terminated fatally because there was a calculus in the right ureter also, and he thought he overlooked it because he examined with the left finger.

59. PARENCHYMATOUS GOITRE IN A GIRL, AGED SIX YEARS.

By ERNEST JONES, M.D.

History.—Swelling in neck came gradually six months ago. No pain on pressure. No other case in family.

Physical signs.—A central tumour with bilateral extensions, size of a small Tangerine orange, moderately soft consistence, uniform feel. Movement on deglutition. Situation rather higher than that usual with thyroid tumours. No evidence of Graves's syndrome present.

(*Exhibited February the* 16*th,* 1906.)

60. A CASE OF INFANTILE SCORBUTUS.

By GEORGE CARPENTER, M.D.

The infant, aged 10 months, is slightly rickety, a trifle anæmic, and shows hæmorrhagic gingivitis, subperiosteal hæmorrhage of the left tibia and hæmaturia. The urine contains albumin and blood-casts, but is free from other casts. It was fed on Allenbury's and other patent foods.

(*Exhibited February the* 16*th,* 1906.)

61. STRYCHNINE IN THE TREATMENT OF CHOREA MINOR: A PRELIMINARY COMMUNICATION.

By WILLIAM EWART, M.D.

Had the writer been aware that strychnine had been long ago extensively tried, and since then completely given up, he might not have undertaken its clinical study. As a fact, Trousseau's systematic treatment was planned on the idea that strychnine was, as it were, a specific, and that its administration should be pushed till toxic symptoms set in. The present method differs from this in both particulars. Strychnine is to be prescribed merely as a nervine and general tonic, and though it has to be pushed, any approach to toxic symptoms is carefully avoided. This is best secured by the combined influence of ammonium bromide. Obvious benefit has been obtained in children of six to twelve years of age from doses of the liquor varying from ♏v to ♏x administered three times daily, and guarded in this way by bromide.

The indications of treatment which had been previously followed has not been given up.

(1) The anti-rheumatic indication is still met by salicylate.

(2) The tendency to valvulitis is still combated by iodide of potassium.

(3) The spasmodic agitation is quieted by such remedies as bromide, hyocyamus, and syrup of red poppies, and lastly,

(4) The wear and tear of the nervous system is partly repaired by nerve food, such as the glycerophosphates and the hypophosphites.

The strychnine is now administered side by side with these remedies with manifest advantage. But no rapid cure is to be expected from it, and it is doubtful whether the total duration of the attack has been appreciably reduced. Convalescence was, however, greatly shortened owing to the excellent condition secured before the total cessation of the spasms.

(*Read March the* 16*th*, 1906.)

Discussion.

The Chairman (Dr. G. A. Sutherland) expressed the Society's gratitude to Dr. Ewart for bringing the matter forward. He said the author seemed to imply that the stimulating treatment of chorea was not very common. He (Dr. Sutherland) thought a large number of cases were treated by stimulants rather than by sedatives, and his method had been to give as full a diet as possible, complete rest in bed, and where there was weakness brandy in fairly full doses. He also usually gave nux vomica, but not strychnia. Many cases showed much weakness, and the muscular power was usually found to be diminished. He desired to know whether Dr. Ewart gave all the drugs he mentioned at the same time, namely, sedatives, anti-rheumatics, builders-up, such as glycero-phosphates and strychnia. Where so many drugs were given it was difficult to know what part each played in the treatment.

Dr. Leonard Guthrie said he had only had experience of strychnia as a general tonic. Dr. Ewart's case improved after four and a half weeks' treatment, and most cases improved in that time without strychnia, but with rest in bed and good food. He had never advocated large doses of alcohol indiscriminately, but he had found it necessary in severe asthenic cases. He (Dr. Guthrie) thought strychnia might be of advantage where too many sedatives had been given. He would like to hear whether Dr. Ewart had used strychnia in the extremely severe cases which lingered on for months and defied treatment.

Mr. R. Clement Lucas said the point of interest to the surgeon was that some cases of chorea occurred immediately after an accident, without there being any evidence of anything amiss before and without evidence during the attack of any rheumatism, either in the heart or the joints. Were all cases of chorea dependent on the same cause? If a patient had a slight dose of the diplococcus (which most people now believe to be the cause of rheumatism) and an accident occurred upon that, did chorea develop? Either there must be more than one cause for chorea, or there must be some explanation of the cases which occur immediately after injury.

Mr. Milner Burgess (Harlesden) asked whether Dr. Ewart dieted the children in any way.

Dr. F. J. Poynton said he had always had the best results in very bad cases of chorea from the administration of chloral.

Dr. Eric Pritchard asked whether Dr. Ewart thought there might be an antagonism between the therapeutic action of strychnia, which was to cause tonic contractions, and the clonic contractions characteristic of chorea, and that this might explain the reported good results of the treatment.

Dr William Ewart, in reply, said he would like the opinion of neurologists on the point raised by Dr. Pritchard. He had not gone deeply into the theory of the matter, but gave the strychnia under the idea that the chorea was an infection. The most telling cases were the paralytic ones. In other cases there were spasms, in consequence of the paralysis of the controlling centres, and anything which pulled the controlling centres together, such as a tonic, would improve the spasms. He had very little experience of surgical cases. He would be inclined to try chloral in bad cases. His own treatment for many years in bad cases had been by sedatives, but he had not had any case of violent chorea since he began to use strychnine. In a case of alarmingly bad convulsions threatening life, strychnia would have a secondary place, but he would administer it in a modified form. He recently had in his ward a case of epilepsy which came from Southampton in a marked condition of intellectual bromism. There was very great sluggishness in answering questions. He administered strychnia, and although the fits were not cured, the patient was much smarter in every way. The result was due to the strychnia, because no other agent was employed. In the treatment of epilepsy he thought the right thing was not to push the dose, but to see how low the dose could be got to derive advantage from the strychnine and yet not expose the man to a continuance or aggravation of his fits. He was glad to hear that Dr. Sutherland gave nux vomica, and he would be glad if strychnia could be given a similar trial at his hands. Though the strychnia did not seem to abbreviate the duration of the chorea there was a marked improvement in the patients generally. Under strychnine his opinion was that the patients were better at the end of six weeks than without it. He gave the patients easily assimilable food.

62. CASE OF SYMMETRICAL DIMPLING.

By ERIC PRITCHARD, M.D.

This is a case of symmetrical dimpling on the dorsal aspects of the extremities of the two acromial processes in a girl, aged 8 years. The condition was discovered at the time of birth and, therefore, was clearly congenital. The dimpling is of an extreme nature, especially on the right shoulder, and in character resembles the dimpling which is seen as a more or less physiological condition over other bony prominences, such as the mental eminence of the inferior maxillary bone, at the elbow and at the wrist, especially in fat children. Whether the dimpling in the case exhibited represents an exaggerated form of what is a physiological condition, or whether it is to be included in the category of stigmata of degeneration, I am not prepared to say. In favour of the former hypothesis is the fact that the child at the time of birth was particularly plump, not to say fat; and with an extensive deposition of fat round a point of relative fixation of the skin, such as may occur over bony eminences subject to pressure and friction it is conceivable that the surrounding skin may have been sufficiently elevated to cause the dimpling exhibited. In favour of the second view, namely, that the dimpling is to be regarded as a stigma of degeneration, the following evidence may be adduced. The father is an alcoholic; three of the patient's brothers died in early childhood of diseases referable to the nervous system, while a fourth, and only surviving one, is at this present time a helpless paralytic. The patient herself exhibits no marked signs of mental or physical inferiority, although there is some slight want of symmetry in the formation of the hard palate. She is stated by the mother to be of a very obstinate disposition, and to be subject to occasional outbursts of temper of a very violent kind.

Mr. Howell Evans tells me that in a case of "symmetrical bilateral branchial fistulæ with symmetrical bilateral helical fistulæ and mis-shapen aural pinnæ" which he showed to this Society on January the 19th, 1904, there were also symmetrical dimplings over the acromial processes. As Mr. Evans has not seen my case, and I did not see his, I am unable to say whether there is any parallelism between the two cases, but, to say the least, the evidence is suggestive that the dimpling in the case I have shown represents some form of misdirected development.

(*Exhibited March the* 16*th*, 1906.)

Discussion.

The Chairman (Dr. G. A. Sutherland) said the notable point was the position of the dimpling. He did not remember seeing a case in which the dimplings were in the same position. As it was close to a bony point, there may have been some intra-uterine pressure.

Dr. Edmund Cautley said he believed the general explanation advanced was that such dimpling was the result of amniotic adhesions. He thought the case belonged to the same group as those in which there was a constriction surrounding the whole limb, advancing sometimes to amputation of the limb. Very little proof of the theory existed.

Mr. R. Clement Lucas said the interest of such cases lay in the family history, showing that there was a decided mental and nervous defect associated with the apparent accidents to which reference had been made, such as amputations, adhesions, and dimplings in certain situations. There was defective development probably very early in the embryonic state.

63. A CASE OF ACHONDROPLASIA.

By KENNETH R. HAY, M.B.

(Introduced.)

J. K—, aged 2 years, may be considered, I think, to be a typical example of the condition called "achondroplasia." He is the elder of two children. The younger is aged 4 months, and is, to all appearances, normally shaped. The father suffers from phthisis, and has been subject to fits, probably epileptic in nature. The mother is not a robust woman, but says she has always enjoyed good health. There is no history of syphilis.

At the time of the child's birth the mother was in labour about twenty-four hours, and delivery was effected by instruments. From the first the child was, according to the mother, always weak and ailing, and the most noticeable features about him were the large size of his head and the stunted character of his limbs. He was fed on the breast up to the age of ten months, and since then has had the usual diet of a child of his age.

His intelligence is, perhaps, rather below the normal for a child of his age. He can pronounce the words "daddy" and "mummy," but otherwise does not talk. He is inclined to be irritable and to resent any interference. He can stand and also walk a little with aid, but is very unsteady. In the standing position the typical lumbar curve is well seen.

The head is decidedly large, and the frontal bones prominent. The root of the nose is depressed. The anterior fontanelle is open. The circumference measures 20½ inches.

The body is long relatively to the limbs, and the chest rather narrowed. There is slight beading of the costochondral joints of the ribs. The heart and lungs are normal, and the sexual organs well developed.

The limbs are shortened in a characteristic manner. The hands and fingers are dumpy. The humerus and ulna each measure 3¾ inches. The femur measure 4¾ inches, and the tibia 5 inches. The bones feel thick and hard, and are not apparently curved. (*Exhibited March the 16th,* 1906.)

Discussion.

The Chairman (**Dr. G. A. Sutherland**) asked whether there had been any similar condition in any other member of the family.

Dr. F. J. Poynton said that he believed it true that achondroplasic cows threw not infrequently cretinous calves, and for that reason it was thought that cretinism and achondroplasia were associated, and thyroid tried but with indifferent success.

Dr. Edmund Cautley said it was interesting to see a case at such an early age. In reference to Dr. Poynton's remarks, he did not consider that thyroid treatment could be expected to do good. Post-mortem evidence had shown no affection of the thyroid, nor of any disease existing at the time of death, but only the remains of an early intra-uterine condition. The effects were mainly evident in the long bones. Sometimes, also, there were softening and bending of bones, or rachitic conditions might be associated. The head always looked disproportionately large. The children were difficult to rear, but if they survived they went on to normal development, except that they had short arms and legs. As a rule they were mentally unaffected. When mental backwardness was noticed, it was often traceable to their having been kept away from school on account of their deformity.

Mr. George Pernet said the deformity was apparently worshipped by the ancient Egyptians, under the name of Ptah-Seker-Asar, the triune god of the resurrection. He exhibited pictures with which he had illustrated an article contributed to 'The British Journal of Children's Diseases,'* from statuettes which could be seen in the British and most other great museums.

Mr. Kenneth R. Hay (introduced), in reply, said there was only one other child, and there was no sign of disease in it. He had not yet tried thyroid extract. The child had only been under observation three weeks, but his reading did not lead him to expect any good from thyroid.

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* "A Note on the Antiquity of Achondroplasia," vol. i, 1904.

64. A CASE OF ERB'S PARALYSIS IN A CHILD AGED FOUR AND A HALF MONTHS, INVOLVING THE FIFTH, SIXTH, AND SEVENTH CERVICAL NERVES.

By HUGH LETT, Ch.B.

(*Exhibited March the* 16*th*, 1906.)

Discussion.

Dr. Robert Hutchison said the prognosis of such cases was especially interesting. Erb's paralysis was very rare in grown-up persons, and the fair inference was that the majority of such cases were recovered from if left alone. He selected a dozen cases from his out-patients during the last five years and followed them up, and was surprised how, even after a long time, improvement still went on. He would say that certainly operation was not justified at such an early stage as in the patient now under discussion. Though the progress might be slow he believed the majority of cases got well without operation. He had often put on a splint in the early stage, but not for long, as he feared atrophy of the muscles might ensue. Massage was very difficult to carry out in the small muscles of a baby, and though electricity was useful theoretically, practically for an infant it was out of the question.

Dr. F. J. Poynton agreed with Dr. Hutchison's remarks, and said the child would in two years' time probably have a good arm, certainly a better one than if operation were done. He would not even advocate a splint for it, but steady rubbing and moving about.

Mr. Lockhart Mummery agreed with Dr. Hutchison's remarks. He had a case at the North-Eastern Hospital with more paralysis than in the present instance. It had been under treatment four months, and the improvement was steady and appeared to be still going on. He would not operate in similar cases until a very much longer period had elapsed. He would concentrate attention on the prevention of adhesions in the joint, which were sometimes severe. In his case the shoulder-joint adhesions were so great that they had to be broken down, and there was still a tendency for them to form. It was important to see the child occasionally and move the joints, and also instruct the mother to do so.

Mr. Milner Burgess (Harlesden) said his own experience had been that such cases recovered slowly; he had not seen bad ultimate effects.

Dr. Edmund Cautley thought the speakers took too optimistic a view of such cases. He knew one child which was not cured by the eleventh year. Undoubtedly most of them slowly recovered, unless they died of intercurrent disease.

Dr. Leonard Guthrie agreed with Dr. Cautley, as he had also seen cases in which there was no improvement. The prognosis depended on the lesion. If the fifth and sixth nerves were broken across it was difficult to see how regeneration could take place spontaneously. If the nerves were simply bruised, subsequent improvement could be easily understood.

Mr. Hugh Lett, in reply, said he had no intention of advising operation in the present case as the child was improving, as did most of the cases. But he had seen a boy, aged 15 years, who had not recovered. If operation were desirable, apparently the sooner it was done after a certain age the more rapid was the recovery. Massage was carried out at the hospital twice weekly, and the mother was instructed to rub and move the part systematically. He did not propose to leave off the splint yet as there was still a tendency to dropped wrist.

65. A CASE OF PRECOCIOUS DEVELOPMENT.

By HUGH LETT, Ch.B.

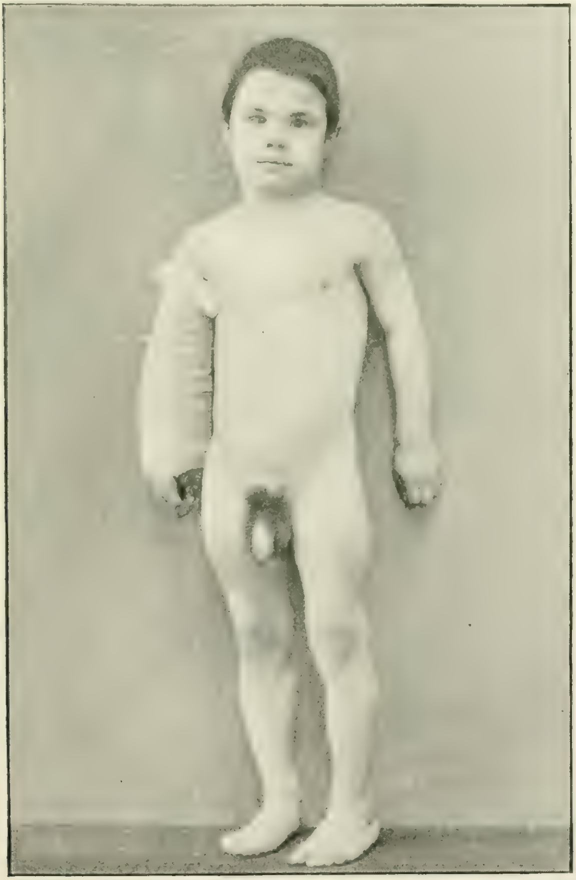
This boy, aged 4 years, had been rather backward until two years old, the pubic hair then appeared, and development took place with extreme rapidity. There was a remarkable growth of hair all over the body, especially over the pubes and in the lumbar region. The penis and testicles were large, and resembled those of a young adult. His voice was deep, and was like a bass voice not yet under full control. His muscular development was remarkable, especially in the thighs, and he was possessed of proportionately great physical strength.

A skiagram of the wrist showed ossification taking place in all the bones of the carpus, and one of the knee presented the appearance of the joint of a boy seventeen or eighteen years old.

No enlargement of the thyroid or supra-renal bodies could be made out.

Intellectually he was bright, but had a terrible temper, had no fits, and was not subject to headaches. His appetite was voracious.

Fig. 9.



There was nocturnal enuresis, which was getting worse. He had frequent erections, and nocturnal emissions during the last three or four months.

He was 3 feet 7½ inches in height, and weighed 3 st. 12 lbs.

(*Exhibited March the* 16*th*, 1906.)

Discussion.

Mr. R. Clement Lucas expressed the indebtedness of the Society to Mr. Lett for bringing forward the case. The subject had not yet been fully worked out. Cases of precocity were rarer in males than in females. He published a case of the latter in the Clinical Society's 'Transactions,' the subject being a girl, seven years of age, who was under his care at the Evelina Hospital. She had well-developed breasts, mons veneris, and long hair on the pubes. The labia were also well developed, and she menstruated at intervals. There was in that case a solid tumour of the ovary, and after that tumour had been removed the abnormal characteristics subsided, except the hair on the pubes.

Dr. G. E. Shuttleworth (Richmond) asked if there was any family history which threw any light on that extraordinary case. He understood the father to be dead, and he appeared to have been ill at the time of the conception of the child.

Dr. F. Parkes Weber said that tumours associated with precocious development were not in every case hypernephromata. Sometimes there were tumours in the ovaries (of which Mr. Lucas had narrated an excellent example) or in the testes. Still, the possibility of the occurrence of hypernephromata in the testes or ovaries, as well as in the adrenal bodies themselves, and in the kidneys and liver, must be borne in mind. Analogous to Mr. Clement Lucas's case in a female, a remarkable case in a male had been described in which extraordinary precocious development had been associated with a tumour of the testis (left). After the removal of the latter the boy became child-like again and his precocious beard fell out (see E. Sacchi, 'Rivista Sperimentale di Freniatria,' Reggio, 1895, vol. xxi, p. 149). He (Dr. Weber) thought that cases of precocious development (sexual and general) were not all connected with tumours. An alternative way of looking at such cases was to regard them as representing a reversion to a lower type of animal, in which development occurred much more rapidly than in the human being. Primitive prehistoric man probably had to develop rapidly in order to survive, though the individual who developed slowly had ultimately shown themselves fittest to survive. In the growth of complex organisms there was a balance in the progress of development in which all the organs had to take their share; and it was easy to imagine that in some cases the balance would be thrown out more readily than in others. This explained why hypernephroma was not always associated with any extraordinary precocity of development. In fact, in some cases the tendency to normal growth of the body was too strong to be upset by abnormality in the suprarenal cortical parenchyma.

Mr. Hugh Lett, in reply, said Mr. Lucas's case was extremely interesting. Suprarenal tumours were found occasionally in the broad ligament of the uterus, and the tumour in this case might possibly have been of the same nature. The mother said the father had considerable sexual development, and had very frequent coitus. He was paralysed on one side when the child was conceived, and subsequently became paralysed on the other side and died. It was difficult to give a prognosis. Precocious development was not associated with suprarenal tumours only, in a few cases tumours of the pineal gland had been found.

66. A CASE OF CONGENITAL COLOBOMA OF THE EYELID.

By SYDNEY STEPHENSON, C.M.

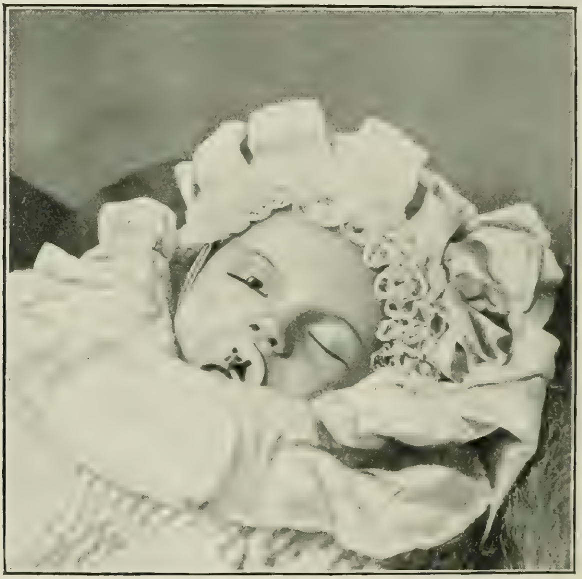
William W—, aged 3 months, was brought to the eye department of the Evelina Hospital on March the 6th, 1906, on account of a deformity of the right upper lid, present since birth.

Upon examination, a quadrangular gap was seen in the right upper eyelid. It involved the inner half of the lid with the exception of a small nodule at the inner end, and extended about halfway from the orbital to the palpebral margin (Fig. 10). The puncta lacrymalia were present, the upper one being situated on the nodule mentioned above. When the baby shuts his eyes, the right cornea and pupil can be seen through the gap in the eyelid, thus producing a somewhat weird effect. There is no particular thickening of the palpebral conjunctiva, no lipo-dermoid of the conjunctiva, no dermoid of the cornea, and no coloboma of the iris. The baby, who has a small fovea sacralis, presents a tiny supernumerary auricle in front of his right ear.

The patient is the fifth child in the family, none of the other members of which have any deformity of the eye or its appendages. The father is stated to have a small nodule in front of his left ear.

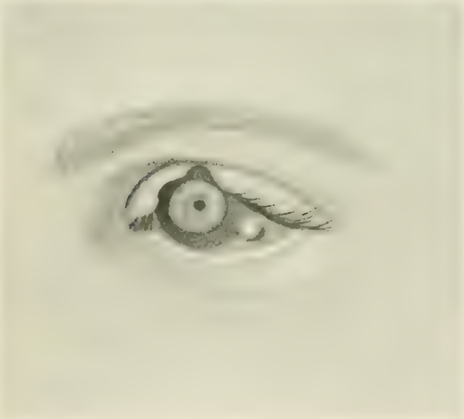
Remarks.—Congenital coloboma of the eyelid is one of the rarer malformations of the ocular appendages. Thus, Dor and Nicolin ('Revue générale d'ophtalmologie,' December the 31st, 1888) collected forty-six cases from the literature. In twenty-seven of these cases the coloboma affected a single eyelid; in two both eyelids of one eye; in sixteen the upper and lower lid of one eye; and in one case all four eyelids. The deformity of the eyelid was associated in fourteen instances with hare-lip, with or without extensive cleft-palate, in twelve with oblique fissure of the face, and once with coloboma of the iris. The authors named trace coloboma to an interference with the development of the eyelids by imperfect closure of the oblique facial fissure. On the other hand, certain writers, particularly van Duyse, believe that the condition is connected with amniotic adhesions.

Fig. 10.



There is a point of some practical interest connected with colobomata of the eyelids, namely, how far such a deformity predisposes to ulceration of the cornea. It seems probable that exposure of the cornea, as in the present case, may lead to slight injuries and to consequent ulceration of a more or less destructive character. I know of no evidence upon the point, but the sketch (Fig. 11) is of such a case in a young child, who was under my care at the Evelina Hospital a few years ago. There was a double coloboma of the left upper lid, and the child was admitted suffering from severe and perforative ulceration of the corresponding cornea. If, as I suspect, the connection between a gap in the eyelid, on the one hand, and an ulceration of the cornea on the other, be a tolerably close one, we are furnished with an additional reason for closing the coloboma by surgical means at the earliest possible moment. (*Exhibited March the* 16*th*, 1906.)

FIG. 11.



Discussion.

Mr. R. Clement Lucas said the question which interested him was the possibility of curing the condition illustrated by means of operation. In the present case he thought it would be wise to persuade the mother to have the child operated upon early, to bring about protection for the cornea, and so prevent the disastrous results which might follow from exposure.

Mr. Sydney Stephenson, in reply, referred to the explanation of this congenital anomaly. The eyelids were supposed to grow out as two fleshy folds, which met together at an early period of intra-uterine life; they were not developed from two portions. The currently accepted theory was that coloboma was connected in some way with the persistence of the oblique facial fissure, and some cases had been recorded in which those two malformations were met with in the same patient. But that did not seem to be a satisfactory explanation. To assume that the persistence of the oblique facial fissure interfered mechanically with the development of the eyelids was a thing which was easier to conceive than to comprehend. He suggested that our knowledge of the embryological development of the eyelid was not complete, and that the eyelid might, after all, develop from two distinct centres, which would make possible an explanation of such malformations as he now showed. He exhibited two illustrations bearing on the subject.

67. A CASE OF CONGENITAL DEFORMITY OF THE HANDS.

By H. S. CLOGG, M.S.

The infant, aged 4 months, is one of twins, the other child being perfectly well formed. There is nothing in the family history of any importance. The period of gestation and the labour were normal; delivery took place at the ninth month.

The *right hand* is shortened and comparatively broadened. The terminal phalanges of all the digits are stunted, and it appears that the bony element is suppressed, there being only the soft tissues present. The nails are very imperfectly formed. Some power of flexion appears present in the terminal stump.

The *left hand:* the fingers of this hand are curiously bound together at their distal extremities. The proximal portions of these are free from one another, and the webs are not unduly prolonged down upon the inter-digital clefts. The distal halves of the digits are fused together apparently by soft tissues only, and overlapped in a curious way. On the flexor aspect of these fused digits there is a distinct groove suggesting a constriction by a band; this is especially evident on the flexor aspect of the second finger. The terminal phalanges are again very imperfectly formed, and it would appear that the bony element in each was absent. The nails are very rudimentary as in the right hand. Flexion of the fingers, as a whole, is possible, the amount of movement in each finger cannot be estimated on account of the fusion. The thumb is free, but the terminal phalanx is suppressed, apparently by absence of its bony element. The child is well formed in other respects.

(*Exhibited March the* 16*th*, 1906.)

68. TUMOUR OF THE PONS VAROLII.

By C. O. HAWTHORNE, M.D.

Patient, a boy, aged 8 years, brought to hospital on October the 28th, 1905, because he "cannot sit still, and sometimes falls off his chair."

The most conspicuous symptoms as seen in the Out-Patient Department were: (1) A constant series of jerks and spasms mainly affecting the trunk, and (2) extreme retraction of the head, but without rigidity of the post-occipital muscles.

There was considerable optic neuritis in each eye, and the knee-jerks were very marked, but there was no ankle-clonus, and the plantar response on each side was of the flexor type.

During residence there was occasional vomiting, and the speech became very slurred and indistinct, but the most striking feature of the case was the extreme retraction of the head with more or less opisthotonos. These conditions reached their highest degree when the patient was sleeping or lying at rest. On the other hand, when roused to sit up, the spine was readily straightened and the head could always be flexed so as to make the chin rest on the sternum. At no time were there any localised paralyses, ocular or otherwise.

The history included attacks of pain in the head, sometimes attended with vomiting, and extending over two months previous to admission into hospital. The boy had fallen when walking on several occasions, and once or twice had fallen off a chair, and even out of bed. No history of injury to the head. (*Exhibited March the* 16*th*, 1906.)

69. SPECIMENS FROM A CASE OF SMALL WHITE KIDNEY (CHRONIC INTERSTITIAL NEPHRITIS).

By C. O. HAWTHORNE, M.D.

Patient, a girl, aged 12 years, admitted into hospital on December the 30th, 1905, complaining of pain in the head, occasional vomiting, and loss of flesh.

Examination showed albuminuria (5 in 1000) and tube casts, hypertrophy of the left ventricle and marked neuro-retinitis with hæmorrhages (bilateral); during residence there was abundant hæmaturia.

History of scarlet fever and diphtheria at five years of age; report from hospital says, "No complications of any kind, no dropsy or albuminuria."

There is nothing in the family history or otherwise to suggest specific disease.

Post mortem.—Some degree of purulent peritonitis. Hypertrophy of left ventricle of heart; weight of heart 6½ ounces. Left kidney 1½ ounces; capsule strips readily and leaves a pale smooth surface; section surface shows very narrow cortex—generally pale with a few mottled and congested patches. The microscopic changes are those of chronic interstitial nephritis. Right kidney weighs 3½ ounces, is otherwise similar to left.

(*Exhibited March the* 16*th*, 1906.)

70. A CASE OF ACNE SCROPHULOSORUM.

By F. J. POYNTON, M.D.

A delicate female child, aged $5\frac{6}{12}$ years, with large cervical glands and a history of tuberculosis on the mother's side.

The rash commenced in June, 1905, upon the arms and legs, in the form of discrete papules, which at first itched very slightly. Since that time these patches have been coming out in crops, some of them appearing on the trunk and an occasional one upon the face.

The papules vary in size, reaching that of a large pin's head; their development is slow, and when fully formed they show a bright red areola around a central scale. As they fade the colour becomes paler and on the legs more purple. Finally, in the centre there is left a slight depression with a thin scar. Under treatment with cod-liver oil and simple external applications there has been slight improvement, but the eruption is still appearing on the thighs and arms, although disappearing from the trunk. The opsonic index has not yet been investigated.

(*Exhibited March the* 16*th,* 1906.)

71. A CASE OF CONGENITAL UNILOCULAR CYST OF THE NECK.

By DOUGLAS DREW, B.S.

(*Exhibited March the* 16*th,* 1906.)

72. CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By EDMUND CAUTLEY, M.D.

I HAVE brought this specimen and the notes of the case before the Society to illustrate certain points to which I shall refer shortly.

The patient was under my care in the Metropolitan Hospital from December the 14th to the 22nd, 1905, and died from asthenia at the age of ten weeks, weighing 3 lbs. 4 oz. four days before death.

She was one of twins, born about three weeks prematurely. The other twin had died. Two elder children were healthy.

She was breast-fed and, though small at birth, seemed healthy for three weeks. Then vomiting began, at first usually after feeding. During the week before admission she had only vomited about once a day, bringing up a large quantity.

Constipation had existed since birth, the stools being small. The child had been given a rectal injection every three days. A tarry stool was passed on the day before admission. She was greatly wasted, weighing 3 lbs. 7 oz. The fontanelle was depressed; pulse 120, small; cry vigorous. The stomach was dilated, reaching almost to the umbilicus. A moderate peristaltic wave could be seen. The pylorus was not palpable, probably being situated beneath the liver, which reached to the level of the umbilicus.

The vomiting ceased when the child was put on whey. At first 1 drachm was given every quarter of an hour and gradually it was increased to 1 ounce every hour. Peptonised milk and water gave rise to more vomiting on the 17th and 18th, 2 to 3 ounces being brought up at once on the latter day. After that whey was given and the vomiting ceased until the 21st, the day before death, when it became characteristic and blood-stained. Throughout the child showed a marked disinclination for food.

A normal stool was passed on the 18th. On other days the stools were small and brownish. On the day before death a stool was passed which contained fæcal matter, of a fair size, greenish yellow in colour. For the last six days of life the temperature remained consistently below 95° F.

On account of the asthenia, the wasting, and the persistently subnormal temperature, operative interference was out of the question.

Post mortem.—The stomach was as large as that of a child, aged 5 months, weighing 11 lbs., who had died the same day from broncho-pneumonia. The pylorus showed the characteristic hypertrophy. It was $\frac{5}{8}$ths of an inch long, and the amount of circular muscle was more than five times as great as that in the other child. The mucous membrane was thrown into two folds. A good-sized probe passed easily through the pylorus, but the canal was minute when compared with that of the other child. The intestines contained a small amount of normal fæces.

This case is very similar in character and course with one I reported previously (‘Reports of the Society for the Study of Disease in Children,’ 1904, vol. iv, p. 39). In each of them the pylorus was partially pervious, but apparently not sufficiently to insure satisfactory nutrition, and death resulted from marasmus.

Comments.—First, I wish to draw attention to the fact that constipation was present from birth and that vomiting did not begin until the age of three weeks. It is much more usual for vomiting to be the first symptom. May not this be taken as evidence of a certain amount of pyloric obstruction at birth? The presence of such an obstruction to the passage of food would lead to constipation, to lack of appetite, and to the gradual development of dilatation and characteristic vomiting. It indicates that the vomiting is the effect of the mechanical obstruction and not due to a pyloric spasm.

Secondly, I wish to point out that the obstruction never became complete and that the intestines contained normal fæces after death. Hence, if a dilute and sufficiently nutritious food could have been given, a food which could pass through the pylorus, the child might have lived. But when one attempted to give a more nutritious food the child again began to vomit.

Thirdly, there was no indication that lavage or antispasmodics were, or could have been likely to prove, of the least value. The vomiting was clearly due to overdistension of the stomach and to no other cause.

Finally, the presence of a tumour must not be relied on for diagnosis. It may be completely hidden, as in this case, under the liver and not be palpable. Peristalsis also was not very characteristic, and the diagnosis was based on the history of the constipation, the vomiting, the dilated stomach, and the wasting.

(*Exhibited March the* 16*th*, 1906.)

Discussion.

Dr. G. A. Sutherland understood Dr. Cautley's remarks to imply that there was no possible treatment of such cases save operation. But it appeared that if the case had been seen earlier there was a possibility of life having been preserved by strict dieting, etc., seeing that it passed a normal motion before death.

Dr. Edmund Cautley, in reply, said that some people maintained they could cure the condition without operation, *e. g.* with infinitesimal doses of opium, or by washing out the stomach. His own experience was that all the cases which were definite and undoubted died if not operated upon. People who said they could cure them without operation did not bring forward specimens removed from babies at an older age. If they were curable in the way mentioned there would surely be instances forthcoming to show the hypertrophy of the pylorus in some of the numerous post mortems made on infants. When not operated upon the babies died in the third or fourth month of life. There was a mild degree of the condition in which some food would pass through the pylorus, and cases were known which lived to five or six years of age, and one until eleven years of age. In the better classes of life, if the cases were seen in the first six weeks and the children were not allowed to waste, they nearly always recovered after operation, He had seen four such cases privately. Three of these were diagnosed in the first four to six weeks of life, were operated upon, did very well, and recovered. They were breast-fed from the commencement, and when that proved unsatisfactory they were carefully fed otherwise, showing that diet had nothing to do with it. The fourth case he had to send into hospital for operation, and after passing through a serious state of marasmus, it recovered. Hospital cases which were operated upon had done badly. Of the last six cases he had seen in hospital, four had been operated upon. One died twenty-four hours after operation from shock; another died three days after operation after apparently doing well, and a little peritonitis was found about the liver and intestines; another lived three or four months afterwards and then died from gastro-enteritis; the fourth was a marasmic child, which lived about two months, he survived one attack of gastro-enteritis, but after another attack he died. The two last he claimed as successful results as regards operation. The specimens from three of these were shown at a previous meeting of the Society. The remaining two were very small and wasted. The diagnosis was not absolutely certain in one of these (*vide* 'Reports,' vol. iv, 1904, p. 39), and the child gained weight under treatment. Then gastro-enteritis developed. The sixth case was the child whose stomach he now showed. Therefore, the prognosis of operation in this condition was the prognosis of the degree of marasmus before operation. He did not think the particular operation was material, but he had a preference for pyloroplasty.

73. A CASE OF MORPHŒA.

By S. J. WAREHAM.

The patient is a little girl, aged 7 years, who came to the out-patient department for some other slight ailment, and in the course of examination a patch of morphœa on the abdomen was noticed.

There is nothing in the family or personal history to throw any light upon the cause; none of the family have had any sort of skin disease, and there is no history of blow, burn, or other injury to the part, or rheumatism, all of which have been given as causes.

The time of onset is indefinite. About nine months ago the mother says she noticed a pimple, and from this pimple the whole patch has spread; there was never any pain, tenderness, or discomfort of any sort from it to suggest a nerve-lesion. The patch has remained much as you see it since first observed by myself.

It is about 1½ inches in diameter, there are no other patches; it is a waxy, dead-white or ivory-like surface, hard, inelastic; the skin cannot be picked up over it; its margins are streaked with vessels, which give it a somewhat lilac tint; it is not anæsthetic.

The only other lesion of skin it simulates is that of anæsthetic leprosy. From this I think it can be readily diagnosed by its being one patch only; it is not anæsthetic; there have never been any symptoms suggestive of nerve-lesion, such as one gets in leprosy; and the colour is too white, leprosy being usually a slight yellow tint.

The distribution of leprosy is not usually on the abdomen. From leucodermia it is easily recognised, in that it is something more than just a loss of pigment from the rete.

My chief reason for showing this case has been to get suggestions as to treatment. There are many treatments, such as drugs and thyroid, which this patient has been taking with doubtful effects; massage is also recommended, and excision has been done by some. Inasmuch as quite a number get well spontaneously it is doubtful as to the efficacy of any one line of treatment.

(*Exhibited April the 20th,* 1906.)

Discussion.

The Chairman (Dr. Porter Parkinson) asked whether any section had been made of the nerves in the affected area, seeing that the disease was considered to be nervous, and if such had been done, had it limited the spread of the disease? If the disease was trophic, one would imagine that subcutaneous section of nerves would prevent its spread.

Mr. S. T. Wareham, in reply, said he was not aware that sections had been done, but thanked the Chairman for his suggestion.

74. A CASE OF PSORIASIS GUTTATA.

By MILNER BURGESS.

The patient, J. B. A—, is aged 6½ years. A paternal aunt died of tuberculosis. On the father and mother's side of the family there was a history of rheumatism, and on the mother's one of "catarrh." His father was treated for syphilis when a young man. Two other children, and no miscarriages.

A few days after birth he had erythema of the buttocks, and subsequently varicella and pertussis.

At five years old, he was seen with slightly raised, brownish red spots, the size of small peas, dry, and desquamating slightly; there were one or two spots on the abdomen, and one or two spots at the back of the right ear; the lesions were distinct, the skin was quite clear between them, and there was no eruption on the elbows or knees. The spots were noticed after the skin had been rubbed with camphorated oil.

At six years old he developed a circular patch at the anus not unlike eczema, and a month later a dry eczema-like patch appeared at the back of the right ear, together with dry pustular spots all over the scalp, forehead, chest, between the scapulæ, and the lower part of the back, and a distinct psoriasis patch on the elbow, which did not appear until ten months after the commencement of the disease.

Last month the large patches of psoriasis were all better on the chest and abdomen, and on the anterior and inner surface of thighs. His back is covered with small patches, the size of small peas, with crusts; there are a few on the scalp and at the back of the ears, and one spot on the forehead near the roots of the hair. The boy is healthy-looking.

He has been treated topically by creolin baths, alkaline baths, Unguentum hydrargyri ammoniati, and Unguentum zinci oxidi, by Unguentum sulphuris to the head and scalp, and by chrysarobin 10 grains to the ounce of liquor gutta percha to the body and extremities, and internally by arsenic. He has been instructed to discard sugar and to avoid much butcher's meat, salt meat, or fish.

In my experience psoriasis guttata is not common in middle-class practice, and I have not seen it develop before in so young a child.

I should like to hear the opinion of members with regard to its pathology, indicating a line of treatment, internal or external, or both. Whether gout, rheumatism, syphilis, or tuberculosis, play any part in its causation, or is it a parasitic disease? Whether diet is or is not important. Whether sea air benefits such cases. Is it contagious? Is the primary lesion in the epidermis, or in the true skin? Is it an hereditary disease?

(*Exhibited April the* 20*th*, 1906.)

Discussion.

Dr. Porter Parkinson pointed out that there was apparently in some cases of psoriasis a connection with rheumatism, as shown by the association of psoriasis and chorea in the same patient in some of the instances he had seen. At the present time he had two children under care suffering from chorea and psoriasis, and he believed he could bring forward notes of three other cases in which the same association was observed.

Mr. R. Clement Lucas said the remark of Dr. Parkinson opened up the question whether one was to consider rheumatic states as due to a particular diplococcus, or whether Dr. Parkinson was speaking only of a tendency for certain individuals to take up that organism. The present bias of the profession was to regard rheumatic disease as due to a diplococcus, and, therefore, it would be interesting to hear whether the Chairman considered that that organism was circulating in the blood or in the system when psoriasis was manifested. Psoriasis was an hereditary condition.

Dr. Parkinson, in answer to Mr. Lucas, said he was aware of the views that some cf the cases of chorea were due to the presence of the organism which was regarded as the cause of acute rheumatism, and he only desired to mention the frequent association of chorea and psoriasis. A large number of skin eruptions were associated with rheumatism, and he did not know whether it was considered that the organism of rheumatism was present in them. The same might apply to psoriasis, though the latter being a chronic disease it would not be so likely to appear as in an acute one like rheumatism.

Dr. Edmund Cautley asked if there was any history of acute specific fever, as some cases had definitely followed fevers, and some had been ascribed to vaccination. It was unusual to see the disease at such an early age, yet a case had been recorded occurring as early as the fifth day, and a few in infants, aged three to eighteen months. In a boy, under his care, the rash was so marked that there were spots even on the penis.

Mr. Milner Burgess (Harlesden), in reply, said the disease did not follow any specific fever.

75. A CASE OF SARCOMA OF THE LUNG (?).

By EDMUND CAUTLEY, M.D.

Male, aged 10 years. Early in January had an attack of (?) pneumonia of right lower lobe. Ailing since.

February the 12th.—Physical signs of pleurisy at right base.

February the 16th.—The screen showed a horizontal shadow on the right side, and no movement of the right half of the diaphragm.

February the 23rd.—Explored under an anæsthetic, but no pus found.

March the 6th.—Breath sounds feeble, and note still impaired at right base.

March the 16th.—Has gained about 4 lbs. in weight. Still dulness, deficient air entry, and diminished vocal vibrations over right lower lobe. Signs of a cavity subjacent to right nipple.

March the 23rd.—More extensive signs of breaking down of the lung, but no expectoration. Very deficient movement and air entry.

March the 31st.—Signs of a large cavity over right lower lobe behind.

April the 17th.—The signs of excavation have cleared up. Dulness is more extensive, and extends on to the sternum. Breath sounds are weak from the third to the fifth rib, and almost absent below that in front. Dulness, feeble breath sounds, feeble bronchial breathing, and bronchophony.

For about ten days he has had some vomiting, cough, and slight hæmoptysis. The heart does not appear much displaced. Practically free from fever for seven weeks.

(*Exhibited April the 20th,* 1906.)

Discussion.

Dr. James Carmichael (Edinburgh) asked whether there was any evidence of glandular affection in any part of the child's body, especially in the thorax or axilla.

Dr. Edmund Cautley, in reply, said he had found no evidence of glandular enlargement, but there was a sign of obstruction to venous circulation in the chest, viz. a little fulness above the clavicles, which might be due to the mediastinum, or to mediastinal glands. There was no movement of the diaphragm on the right side.

76. CASE OF CHLOROMA IN A YOUNG CHILD.

By J. McCAW, M.D.

F. J. K—, aged 18 months, was admitted into Belfast Hospital for Sick Children, on October the 30th, 1905, with a history of having been ill for three weeks.

Family history.—Breast-fed for first nine months, then on a mixed diet. Father and mother healthy. No constitutional taint of any kind found.

On admission.—Child appeared well developed and well nourished, but markedly anæmic, and it had a peculiar frog-like appearance due to proptosis, to swelling and ecchymosis of the eyelids, and also to a swelling about the size of a tangerine orange in each temporal fossa. The proptosis and swelling were greater on the right side than on the left. The swellings were hard, dark coloured, and gave no evidence of fluid contents. A tumour, the size of an egg, was situated over the upper part of the sternum similar to those in the temporal fossæ. The glands in the groins were swollen, hard, and black-coloured, but not tender. The axillary glands were slightly enlarged. The lower end of the left femur was swollen, and very tender on movement. The gums were unhealthy, but not swollen or spongy. The heart and lungs were healthy; no enlargement of the liver and spleen was present. The urine was normal, and the bowels were open. The temperature was 99·4° F.

Progress.—The child became gradually worse, and died three days later. No post-mortem examination could be obtained.

Blood examination (by Dr. Beatty, of Belfast, who also read the communication in Dr. McCaw's absence).—Red corpuscles 1,150,000 per c.mm., white corpuscles 16,000 per c.mm., hæmoglobin 34 per cent. Differential count:

Polymorphonuclears . . .	28·8	per cent.
Small lymphocytes . . .	59·0	,,
Large lymphocytes . . .	8·6	,,
Transitional lymphocytes . .	1·2	,,
Myelocytes	2·6	,,
Eosinophiles	0·0	,,
Normoblasts	5·4	,,
Megaloblasts	0·6	,,

Marked variation in size of red cells, varying from 5μ to 11μ. Two fifths were over the average size, and some were undoubted megalocytes. Poikilocytosis was common, and so was polychromasia. Many of the normoblasts were in mitotic division.

(*Read April the* 20*th*, 1906.)

Discussion.

Dr. R. M. Beatty (Belfast), in supplementing Dr. McCaw's notes, said he saw the child in the out-patient department of the Queen Street Hospital, and was much struck by its appearance, which was different from anything he had ever seen before, and to which the term "frog-like" was applicable. The bulging eyes, swellings on the temples, marked anæmia, and blackness round the eyes made a striking picture. He sent it to the ward labelled acute lymphatic leukæmia, but examination of the blood showed that such was not the diagnosis. From the puncture for the blood examination there exuded clear serum. An interesting feature was the black colour of the tumours, or perhaps a greenish black. He was sorry the account was so incomplete, but the child was taken from the hospital and no post-mortem examination could be obtained, so that there was nothing more than the clinical picture to present. It was not like a case of pernicious anæmia, and he thought chloroma was the most likely diagnosis.

Dr. Edmund Cautley said chloroma was so rare that probably no one present had seen a case. He believed only about thirty cases were on record. The present case was peculiarly interesting in that it was diagnosed during life, but it was most unfortunate that the diagnosis could not be confirmed by post-mortem examination. There was not the degree of leucocytosis that one would expect. The characteristic blood change of chloroma is an enormous lymphocytosis, consequently 16,000 white cells did not seem a sufficiently high proportion in a child of this age, and 59 per cent. of small lymphocytes did not indicate a pronounced lymphocytosis. He did not remember that profound anæmia occurred in these cases, or profound changes in the red cells. The child in question was 18 months old, and in a child of that age, ill from any serious disease, the blood conditions reverted to very much what was usual in the earlier stages of existence, so that lymphocytosis was much more readily induced. There was a possibility of the case being due to other form of tumour.

Dr. James Carmichael (Edinburgh) said a case of chloroma occurred in the wards of one of his colleagues in the Children's Hospital, and in that case the facial appearance agreed with Dr. McCaw's case. The tumours were very marked about the region of the eyes and orbits, and there was some peculiar affection of the femoral bones. He had not heard that the patient died or that there had been any post-mortem examination. The blood examination did not reveal quite the same appearance as that described by Dr. McCaw; there was not the same amount of blood degeneration. The general microscopical appearances resembled those of leukæmia. He had not had a similar case under his own observation.

Dr. Beatty (Belfast), in further reply, said no tumour could be discovered in the abdomen by anyone who made the examination. The glands in the groin and axilla were slightly enlarged. He asked members to remember the very short course of the affection. The mother stated that the child was perfectly well three weeks previously. The only other similar case of which he knew anything was Dr. Dunlop's case, of Edinburgh, the prints of which he had seen, and the identity with it was rather striking. He had written to Dr. Dunlop giving a description of the case, and that gentleman agreed that the case was probably chloroma. He joined in lamenting that there was no post-mortem examination.

77. THE TREATMENT OF HERNIA IN CHILDREN, WITH AN ANALYSIS OF THREE HUNDRED AND FIVE CASES OF OPERATION FOR RADICAL CURE.

By ROBERT CAMPBELL, M.B.

Mr. Robert Campbell (Belfast) read a paper on inguinal hernia in children, giving an analysis of 305 operations for radical cure. He said that it was generally admitted that treatment by trusses was uncertain in its results, prolonged, and irksome to the patient; whereas by operation a permanent and complete cure could be obtained within a fortnight. The great advantages of operative treatment being admitted, he said the question then arose, At what age should the operation be performed? He thought the only logical answer to this was, As soon as the patient is presented for treatment, provided the general state of health be satisfactory. Some surgeons who had published articles recently on the subject had advocated treatment by trusses until the age of three or four years. What was the reason for this view? Was it that the pressure of a truss might, at the cost of much trouble to the surgeon, and inconvenience or even suffering to the patient, have a chance of producing a cure? Or was it that these surgeons were restrained by imaginary difficulties and dangers of the operation in infants? He quoted statistics to show that 34 per cent. of his cases were operated on under six months, 44 per cent. under one year, and 77 per cent. under three years. Mr. H. J. Stiles and Dr. E. S. Carmichael had also advocated and practised operating during the first six months of life. He thought that the danger of sepsis in infants had been greatly exaggerated. Only two out of 305 cases had suppurated. He also called attention to the nature of the sac. In not more than 5 per cent. of cases did it communicate with the tunica vaginalis testis. In other words, the common hernia of children was a hernia into a patent funicular process, which was, of course, as much entitled to be described as a congenital hernia as that in which the sac and tunica vaginalis were continuous. In referring to the contents of the sac he said that the frequency with which the cæcum and vermiform appendix descended in children was remarkable. He mentioned two cases in which the appendix was found adherent at its tip to the bottom of the sac ; and another case in which a large Michel's diverticulum was in the same condition. Strangulation was not frequent nor acute in children. He had operated on sixteen cases ; most of these were under one year of age, two being twenty-one and twenty-four days old. In one case the ovary and Fallopian tube were found strangulated and gangrenous in the lower part of the sac, while the bladder and uterus were lying intact in the upper part. His experience of ovarian hernia had led him to the following conclusions : (1) That the aperture in the abdominal wall is larger, rounder, and nearer the middle line than in intestinal herniæ. (2) That strangulation is due to twisting of the broad ligament and not to constriction of the neck of the sac. (3) That the ovary involved tends to become enlarged and cystic. (4) That strangulation in these cases is common. As regards after-treatment his usual plan is to remove stitches on the fifth day and paint the wound with collodion ; the patient, if under two years, is sent home on the sixth day ; if over two years, he is kept until the ninth day. His own experience is that the operation is practically free from risk. The mortality should certainly not exceed ·3 per cent. Radical cure of herniæ in children might, therefore, as regards risk, be classified with the many so-called minor operations. (*Read April the 20th,* 1906.)

Discussion.

Mr. R. Clement Lucas said that Mr. Robert Campbell's most interesting paper showed how much advance had been made not only in surgery but in anatomical exactitude as the result of operative measures. As the author said, it was formerly taught that all those cases in children in which herniæ passed into the scrotum were cases of congenital sac, in which the tunica vaginalis was supposed to be open all the way from the abdomen, so that bowel coming down and passing into the tunica vaginalis would surround the testis. Mr. Campbell showed, as his own experience also showed, that they were almost all cases of funicular hernia, in which that part of the tunica vaginalis only was open which covered over the cord and descended to the top of the testis, where it was closed. In a few cases he had found an opening through which he could get a probe, but in by far the majority of cases, as Mr. Campbell had shown, the condition was funicular hernia only, in which the vaginal process was cut off on the top of the testis, and the whole of that portion could be dissected up and cut away without opening the tunica vaginalis proper. With regard to the age for the operation, he believed Mr. Campbell had progressed with other surgeons in the direction of early operation ; and those ancient dicta with regard to leaving on trusses for so many years must be considered things of the past. But the removal of the sac in infants was a difficult matter, as anyone having much experience of the operation must know, for there was great risk of injuring the vas deferens, as it adhered so closely to the surface of the sac. He, therefore, never entrusted the operation to an inexperienced house-surgeon or dresser, nor even if he had had a fair amount of general experience, for injury to the vas deferens was very likely to jeopardise the usefulness of the testis hereafter. He did not gather what Mr. Campbell's dressings were. Since the paper by Stiles, of Edinburgh, he (Mr. Lucas) had adopted the open method of keeping the child fixed and simply using boric powder. He believed his results had been better with that method than by sealing the wounds, which he considered the next best way. The old gauze dressings were so apt to become infiltrated with urine when they could not be rapidly changed, and the wound was liable to be infected. His present practice was to keep the child fixed and the arms and legs bandaged, and a glass tube placed over the penis. Having closed the wound, he simply covered the part with boric powder. Any little exudation which took place could be easily washed away with antiseptic lotion and more boric powder applied. He believed his results had been better since he adopted Mr. Stiles' open method. He felt personally greatly obliged to Mr. Campbell for bringing forward his interesting series of cases.

Mr. Lockhart Mummery considered that the paper was very interesting, as must be any contribution giving a large series of common operations. It was most helpful to know the exact results of these operations. He had very little to say in the way of criticism, because he agreed with all the propositions advanced. The practice in the North-Eastern Hospital was practically the same as that described by Mr. Campbell. The children were operated upon early, but if possible the operation was deferred until the sixth month. Very young children were given wool trusses, but if the hernia proved unmanageable or gave rise to symptoms, the practice was to operate at once. The delay until six months of age was due to the increased difficulty of the operation in very young children owing to the size of the child, and also because it meant weaning the child, which was often undesirable during the early months. He agreed with Mr. Campbell's method of operating. The only thing which was of importance was the ligature of the sac. At the North-Eastern Hospital, Stiles' method of applying boric powder and no dressing was tried, but it was not successful, two cases suppurating, and consequently the practice got somewhat into disrepute. What was now done there was to use collodion or ordinary gauze dressings, kept on with a bandage, and changed fairly frequently.

Mr. Robert Campbell (Belfast), in reply, said he had been agreeably surprised by the remarks of Mr. Lucas and Mr. Mummery as to the custom of operating prevailing in London. His ideas were derived from the published writings on the subject, but Mr. Lucas and others of the advanced school had not published anything on the matter. Consequently he thought it was the regular practice to wait until the child was four or five years of age. And that seemed to him strange in face of the work of Stiles, published three or four years ago. For dressings he was in the habit of using ordinary sterilised gauze and wool, and the sister in charge of the ward was quite competent to change the dressing when it became soiled, and she was instructed accordingly. He had tried collodion and celloidin, but his objection to them had been that they interfered to some extent with the removal of the sutures afterwards. Stiles' dressing was scarcely described correctly as dusting with boric powder. Stiles advocated putting on an emulsion of iodoform and glycerine, and dusting boric powder over that. When he proposed such a proceeding there was a rebellion in his ward, owing to the dislike among the nurses of the smell of iodoform.

78. A CASE OF GASTRIC ULCER.

By EDMUND CAUTLEY, M.D.

The girl, aged 5 years and 4 months, was admitted into the Belgrave Hospital for Children on March the 20th, 1906, for hæmatemesis. Her past history yielded no information of value, except that she sometimes had pain in the lower part of the abdomen. Until the early morning of the 17th she appeared quite well. She then vomited about a pint of dark clotted blood. Soon after this she passed a tarry stool. On the 18th she vomited about half a pint of blood, brighter in colour than on the previous day, containing clots and mixed with food. On the 19th she passed a constipated, black stool.

On admission she was markedly anæmic. A soft systolic murmur was heard at the apex of the heart. The spleen was palpable, and the liver extended about an inch and a half below the costal margin. As the result of an enema, a black stool was passed on the 22nd. After that there was no indication of intestinal or gastric hæmorrhage.

During the last two weeks she had gained 28 ounces in weight. She was still very anæmic, and the spleen was a little larger.

Blood count.—Erythrocytes 3,512,000, and leucocytes 28,250 per c.mm. Of the white cells 63 per cent. were polymorphonuclear cells, 23 per cent. small lymphocytes, and 6 per cent. large lymphocytes. Only one myelocyte and four normoblasts were found.

Remarks.—The diagnosis was not absolutely certain, but had the attack of hæmorrhage occurred in an older girl, it would have been regarded as due to simple gastric ulcer. The absence of a history of dyspeptic symptoms and pain after food is by no means unusual. The enlargement of the spleen suggested a leukæmic affection, but the differential blood-count did not favour that view.

Three types of gastric ulcer occur in the young. One may be found as the cause of hæmorrhage in the new-born, as early as thirty hours after birth. Tuberculous ulcers form a second variety ; usually they are small, part of a general tuberculosis, causing no symptoms, and only found post-mortem. On the other hand, simple ulcers in children are not rarely associated with tuberculous disease. The simple ulcer, apart from other disease, is decidedly rare in children. Extremely few cases have been recorded between the ages of three and ten years. In a case reported by Chvostek in 1883 symptoms began at four years of age, and death took place fourteen years later. Bechtold ('Jahr. f. Kind.,' 1905, Bd. 60), reported a case in a girl, aged 5 years, fatal from perforation and peritonitis ; Reichelt ('Wien. Med. Presse,' 1905, p. 126), a case in a boy, aged 8 years, who died from peritonitis ; Stowell ('Arch. of Pediat.,' 1905, p. 705), a case in a girl, with two simple ulcers, and death from perforation. Stowell reviews thirty-five cases recorded in infants and children. Cutler ('Review,' 1905, p. 2) reported three recoveries in children, aged 6, 8, and 12 years, and collected twenty-six recorded cases, with autopsies in twenty-four of them. Of these, six were under sixteen days old. Thus, it is clear that, omitting cases in the new-born and tuberculous ulcers, gastric ulcer in children is a very rare affection.

(*Exhibited April the* 20*th*, 1906.)

Postscript.—July 5th : Child seems quite well, and has had no further symptoms, no pain, and no anæmia. The spleen is still distinctly palpable.

Discussion.

Dr. G. A. Sutherland said Dr. Cautley had suggested several diagnoses. Most people had very little experience of gastric ulcer in the child, and he could not recall one in which profuse gastric hæmorrhage was the leading symptom. Most of them came under notice for perforation. One frequently met with a history of gastric hæmorrhage, but it was usually associated with some bleeding from the throat, though the present was clearly not a case of this nature. There were several points against it being due to gastric ulcer in the present instance. Apparently there had been no subsequent symptoms, and Dr. Cautley said nothing about the necessity for subsequent treatment. The colour and physiognomy of the child were suggestive of splenic anæmia, and there was an enlargement of the spleen. Hæmorrhages might occur in that affection, whether it was leukæmia or not. There was no reason why the hæmorrhage should not occur from the stomach, although the quantity of blood lost was striking. But the severity of the hæmorrhage depended largely on the local source. His idea was that the hæmorrhage was due to some form of anæmia.

Dr. Porter Parkinson thought the diagnosis was difficult. In his own case of gastric ulcer in the child the patient only brought up a small quantity of blood at the time of the operation. His experience of hæmatemesis in young children had been in purpura and splenic anæmia. In the latter disease the hæmorrhage occurred at a late stage, and had been constantly repeated until death. The present child had not had any bleeding for some time, and that would be against the view that it was splenic anæmia. In other cases the hæmorrhage might have ceased for some time before the patient got very ill. As the present child seemed to be in a comparatively good state of health, he inclined to gastric ulcer as the diagnosis, rather than splenic anæmia.

Dr. Edmund Cautley, in reply, said he thought Dr. Sutherland had not laid sufficient stress on the fact that the child was gaining weight rapidly, namely 28 ounces in two weeks. He did not think a child with splenic anæmia, sufficiently advanced to produce severe hæmorrhage, would gain weight so rapidly. Moreover, the history of the bleeding was typically that of gastric ulcer. There was severe hæmorrhage on two successive days, followed by the usual tarry stools. If it had occurred in an older girl the diagnosis would not have been questioned. The treatment adopted had been that of ordinary gastric ulcer.

Dr. Sutherland desired to add to his remarks that in reference to gaining weight, it was common to find a gain in weight after a severe loss of blood, and he believed it was the practice to bleed cattle when it was desired to fatten them.

79. ATELECTASIS OF BOTH UPPER LOBES OF THE LUNG.

By EDMUND CAUTLEY, M.D.

The child was aged 31 days, and had had a cough for a fortnight. Its past history was unreliable, but it was said to have had no attacks of lividity.

It had been in the Belgrave Hospital for three days. Nutrition was fairly good; weight 6 lbs. 12 oz. The colour was a little dusky, and the temperature subnormal. On examination of the chest, the resonance was much impaired, and the air-entry very feeble over both upper lobes. The breath-sounds were almost imperceptible. Over the rest of the lung the note was good, and the air-entry normal. No catarrhal sounds were heard.

Since admission to hospital there had been little change. A few crepitations were heard over the left upper lobe, and the air-entry was a little better. Attacks of lividity occurred, and the temperature remained subnormal.

Subsequently the attacks of cyanosis and apnœa became more frequent, and the child died during one of them, about a week later. No autopsy was obtained.

This case is interesting on account of the distribution of the atelectasis. Most commonly atelectasis is symmetrical, affecting the lower lobes and the posterior portions of the upper lobes. The diagnosis was based on the physical signs, attacks of lividity, and continuous subnormal temperature. The prognosis was confirmed by the result.

(*Exhibited April the 20th*, 1906.)

80. PHOTOGRAPH OF A CASE OF (?) TETANY.

By EDMUND HOBHOUSE, M.D.

The child gradually assumed the position depicted after some convulsions, and remained in it for five or six weeks with very slight relaxation. The body was rotated on its long axis. The abdomen was greatly distended, and there was probably congenital dilatation of the colon, but permission was not given to examine the abdomen.

There was no disease of the cerebellum or cerebrum.

(*Exhibited May the 4th*, 1906.)

81. A SPECIMEN OF CONGENITAL PYLORIC STENOSIS.

By EDMUND HOBHOUSE, M.D.

The specimen was removed from a child aged 6 weeks. No operation was done, as the child died the night before it was intended to operate.

(*Exhibited May the 4th*, 1906.)

Discussion.

Dr. Edmund Cautley said he would like to ask how long the symptoms had existed in the case of hypertrophic stenosis of the pylorous. In some of these patients the symptoms came on very acutely. He had recently seen a child who went on perfectly well for three weeks, and then began to vomit slightly, but during the sixth week the symptoms became extremely acute, and the child lost nearly one pound in weight, and in the next three days lost another pound. The symptoms were suggestive of an acute obstruction. He thought it highly probable that such cases might be fairly common, but their causation unrecognised.

Dr. Edmund Hobhouse (Brighton), in responding, said that the symptoms in his case came on gradually. It was sent into hospital as a case of marasmus, but it did not appear to be so. There was some vomiting, which seemed to be somewhat relieved by having the stomach washed out. He could not say exactly when the symptoms first appeared, but he thought they were present more or less from the first week after birth.

82. SPECIMEN OF ACUTE YELLOW ATROPHY OF THE LIVER.

By J. PORTER PARKINSON, M.D.

This specimen was taken from a boy, aged 4 years, who had been very healthy till he suffered from jaundice, which appeared nine days before death. No other symptoms appeared for a week, and then he vomited and complained of headache, and the next day had a convulsion. On admission into the hospital on the eighth day of the jaundice his temperature was normal. Pulse rate 84 to 110, irregular, and respiration rate 28. He was restless and delirious, constantly moving the limbs and body, and protruding and retracting the tongue, which was covered with fur. There were sores on the lips. The jaundice was marked. Urine, sp. gr. 1025, alkaline, no albumin and no sugar, but bile-stained. It was not examined specially for urea, leucin, or tyrosin. There were some petechiæ on the skin. Pupils equal and reacted to light. Abdomen distended and tympanitic. The liver dulness above extended to the fifth rib, nipple line; below it could be felt two fingers' breath below the costal margin. Spleen just palpable. The heart and lungs appeared normal on examination.

After twenty-four hours in the hospital the child died comatose, during the interval having developed a general purpuric rash over the trunk and limbs, and a very rapid irregular pulse and irregular respirations.

At the necropsy purpuric patches were to be seen on all the serous membranes of chest and abdomen. Very slight bronchitis. Heart normal. Six ounces of bile-stained fluid in the abdominal cavity. Liver very pale and bile-stained, giving a canary-yellow colour. Lobulation visible on section, some lobules were more opaque than others, and the blood vessels less distinct than normal. The gall bladder contained thick bile-stained mucus which could not be passed down the duct, though this admitted a knitting needle easily.

A portion of the liver is shown stained with hæmatoxylin, demonstrating the necrosis of liver cells, the persistence of the fibroid network, and the presence of nuclei from the degenerated liver cells.

In a freshly-cut specimen which was stained with Sudan iii, the fatty granules in the cells took the stain strongly, and the granular *débris* also stained red.

(*Exhibited May the 4th,* 1906.)

Discussion.

Mr. George Pernet asked Dr. Parkinson if he had made out any etiology in the case, and if there was any congenital syphilis. He asked the question because acute atrophy of the liver in adults had been traced to acquired syphilis. It was a very interesting point. With regard to the rash, he had observed that hæmorrhagic rashes in children were significant of very serious conditions of the viscera, and they will be found under many conditions. He remembered seeing a case which had been brought to the skin department of his hospital with a large number of hæmorrhagic lesions about the body, and one side of the chest was full of pus. So it was a very important symptom in children when a hæmorrhagic rash was present.

Dr. Porter Parkinson, in reply, thought that a purpuric eruption was a feature of the disease. He had seen it in adults, and he had also seen the same eruption in a post-mortem examination as there was in this child. There was no suggestion of phosphorus poisoning. In regard to syphilis he could not be sure.

83. CONGENITAL LARYNGEAL STRIDOR.

By EDMUND CAUTLEY, M.D.

Congenital laryngeal stridor is an abnormality of respiration which is occasionally present at birth, but more usually is only noticed, or comes on suddenly, during the first few weeks of life. It varies in intensity from a purring, crowing, grunting, or harsh croaking inspiratory sound up to distinct stridor. In severe cases a slight noise may be heard on expiration. Its maximum intensity is at the end of inspiration, and, during excitement, it may end in a crow like that of laryngo-spasmus. It is almost always absent during sleep. It is exaggerated by all the causes of deep breathing, and may be associated with a certain amount of recession of the soft parts at the root of the neck and of the lower ribs. Vocal utterance is clear, the cords are normal, crying and coughing are unaffected, and, as a rule, there is neither dyspnœa nor cyanosis. The two specimens exhibited show the anatomical conditions in this affection and the normal larynx of a child of the same age.

The abnormal specimen was obtained from a child, aged 5 months, who died from acute summer diarrhœa.

History of the case.—Lilian G—, born February the 14th, died August the 14th, 1905. She was an in-patient at the Belgrave Hospital for Children from May the 16th to June the 10th, and again during the last three days of life. She was born at full time, the third child of healthy parentage.

Attacks of inspiratory stridor had occurred since birth. The attacks were severe and the dyspnœa urgent, with recession of the soft parts at the root of the neck and, to a slight extent, of the lower ribs. Her general nutrition was fairly good, but she proved unable to digest milk, and had to be fed on condensed milk.

While in hospital the attacks diminished in frequency and severity. The stridor persisted a little during sleep. It was not increased on feeding. She had no nasal discharge, and did not sleep with the mouth open.

On re-admission for summer diarrhœa the stridor was similar in character. The temperature rose to 107° F. before death, which was preceded by convulsions.

Post-mortem.—The epiglottis was found to be folded longitudinally on itself, so that its edges closely approximated. The arytenoid cartilages touched one another, the aryepiglottic folds were closely approximated, and the superior orifice of the larynx was consequently much narrowed. The vocal cords were normal, and the rima glottidis uncontracted.

Reference to the diagrams of the normal and abnormal larynx show very clearly the characteristic anatomical change.

In the few fatal cases on record an anatomical state of the superior largngeal orifice has been found similar to that in the present case.

The infant's larynx is normally soft and collapsible, the epiglottis is gutter-shaped, and the ary-epiglottic folds are drawn together, narrowing the orifice. On these grounds

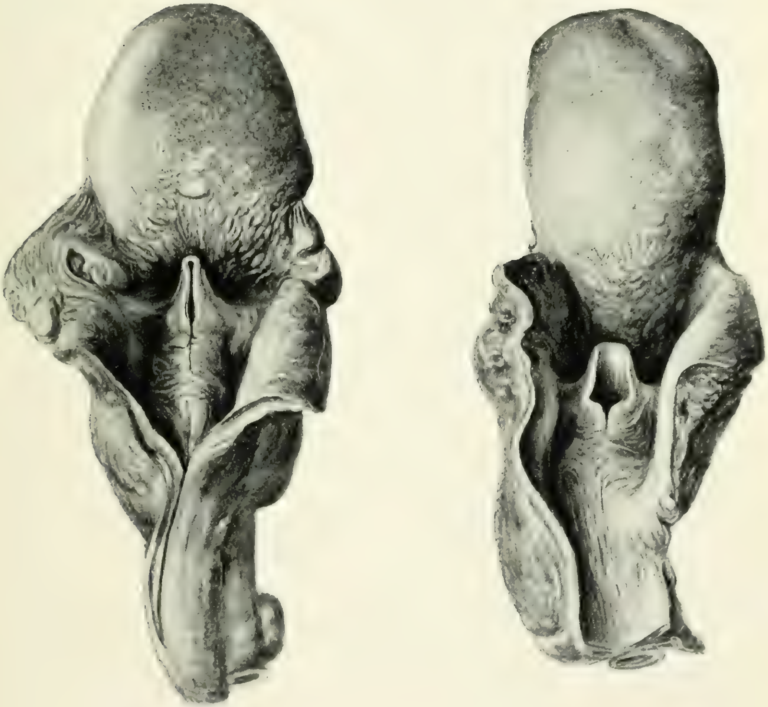


FIG. 1. FIG. 2.

Dr. Edmund Cautley's Case of Congenital Laryngeal Stridor.

Adlard & Son, Impr.

a theory has been advanced that the stridor is not due to an anatomical defect, but that it is simply the result of an exaggeration of the normal state dependent on defective co-ordination of the respiratory movements. The supporters of this view regard the condition as a stammering of the respiratory muscles, the sound being due to the imperfect action of the abductors aided by the soft yielding character of the structures forming the superior orifice of the larynx. Similar stridor may occur during recovery from chloroform narcosis, during excitement, and on sudden deep inspiration. Hence, it can occur independently of anatomical deformity. On the other hand, definite anatomical peculiarities have been found in all the post-mortem examinations, even at the early age of the third month, and there is no evidence of inco-ordination elsewhere.

Possibly both anatomical and nerve factors are concerned, either independently or conjointly. The persistent cases are probably due to anatomical deformity, the stridor being exaggerated by inco-ordination. Thus, we find that the anatomical peculiarity may persist long after the stridor ceases, and the stridor is liable to recur during excitement in later childhood. Until we find typical cases of the affection exhibiting no anatomical change after death, we must regard the inco-ordination theory with suspicion. Congenital laryngeal stridor must not be confused with that resulting from adenoids, nasal obstruction, hyperplasia of the thymus, enlarged mediastinal glands, or mediastinal tumours.

It is rarely fatal, and generally ceases at the end of the first or during the second year of life, perhaps quite suddenly. (*Exhibited May the 4th,* 1906.)

Discussion.

Dr. Edmund Hobhouse (Brighton) said he did not quite see why, if the lesion of the larynx was limited to the epiglottis, there was the stridor, and that was the only thing that puzzled him. He was reminded of a case he had seen in an adult. The epiglottis was precisely in the same condition as the one referred to by Dr. Cautley. It was the case of a man of about forty-five, who was undersized. One certainly did from time to time see an epiglottis in an adult which was practically in the same condition. He would like to know whether Dr. Cautley had any explanation to offer. He himself would not have thought that an epiglottic contraction would have caused stridor.

Dr. W. Bezly Thorne asked whether Dr. Cautley could throw any light on the cause of the change, if there was any, which was associated with the spontaneous cure which such cases appeared to undergo.

Dr. Edmund Cautley, in reply, said the stridor in these cases was purely an inspiratory stridor, and he thought it was produced by the narrowing of the upper orifice of the larynx. The upper orifice in this particular specimen was extremely narrow, and in some instances it had been found narrower even than it was in this. He thought that on deep breathing the epiglottis became probably still more infolded. With regard to the question whether laryngismus stridulus was produced in the same way, he was strongly of opinion that it was. He believed it was due to a spasm of practically the same anatomical area, though in laryngismus he regarded it as pure spasm, while in this affection there was congenital malformation, that was to say, the child was born with an infolded epiglottis. As to why the child progressively got better, he ascribed it simply to the fact that the larynx became larger, and, as it increased, the upper orifice increased. Possibly the muscles attached to the epiglottis became more powerful and kept the epiglottis from becoming infolded on inspiration, for although these cases sometimes got well in that kind of way, and gradually the symptoms ceased, they would find that a similar stridor might be produced in these children at a later period of life, owing to some sudden emotion. In other words, they were still liable to it when they took a deep inspiration. So he thought on the whole the explanation was probably that with the process of growth the larynx became larger and the muscles attached to the epiglottis became stronger. Hence, on inspiration there was not the same amount of narrowing of the upper orifice.

84. ABNORMAL OSSIFICATION OF BOTH PARIETAL BONES.

By EDMUND CAUTLEY, M.D.

It is extremely rare to find abnormal sutures, due to irregular ossification, in both parietal bones. The speci-

Fig. 12.

R.

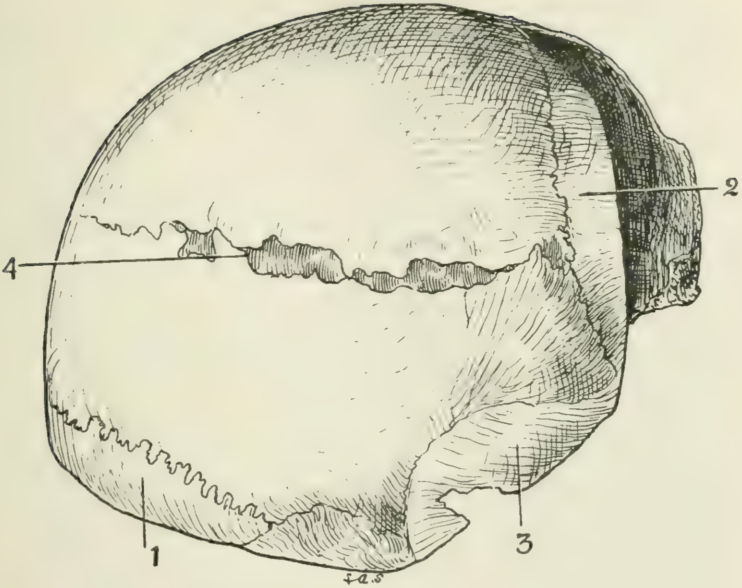
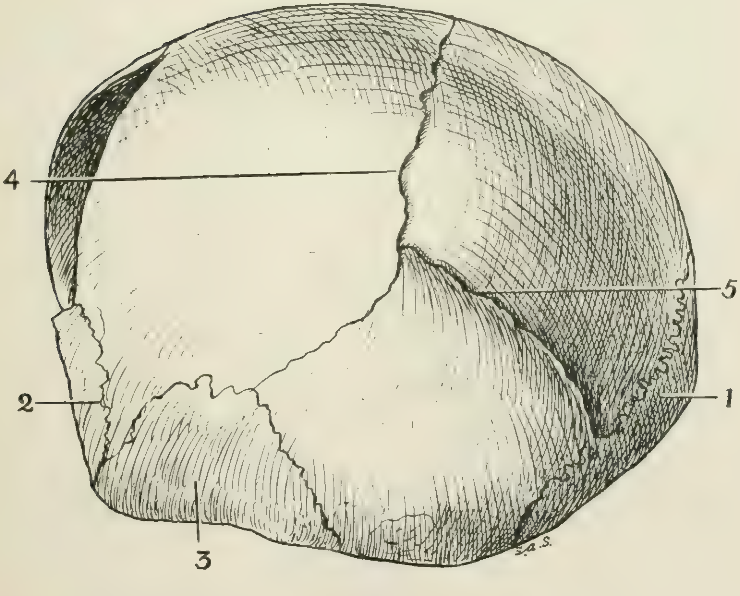


Fig. 13.

L.



men exhibited was obtained from a boy aged 8 months. There was nothing to suggest congenital syphilis at the time, but a year later a third child was brought to me with snuffles and a squamo-pemphigoid rash. In the 'British Medical Journal' for 1899, vol. ii, p. 468, a full description of this case and references to the literature of the subject are given, but as the specimen was not illustrated, and is one peculiarly interesting to this Society, I have brought it forward.

During life the head was large, markedly asymmetrical, with a large anterior fontanelle and soft spots in the regions of the other fontanelles. The sutures on the right side gave the impression of linear cranio-tabes.

The *right parietal* bone (Fig. 12) was divided into two almost equal parts by a well-marked horizontal suture, extending from a point in the sagittal suture, one inch anterior to the posterior fontanelle, to a point in the fronto-parietal suture at the juncture of the middle with the lower third. The posterior half is a simple denticulate suture, while the anterior half is largely membranous. The parietal boss is situated just above and in front of the middle of the suture.

The *left parietal* bone (Fig. 13) was divided into three parts. An abnormal suture, partly dentate and partly squamo-parietal in character, extended vertically downwards from a point almost in the middle of the sagittal suture to the middle of the squamo-parietal suture, dividing the bone into two nearly equal halves. The posterior half was further subdivided into an upper two thirds and a lower one third by a horizontal suture, squamo-parietal and markedly overlapping in character, extending from the abnormal vertical suture to the parieto-occipital suture.

Thus, the right parietal bone consisted of two, and the left parietal bone of three distinct bones.

The cause of this irregular ossification is uncertain. Though there was undoubted syphilitic disease in the third child, it is not clear that this is the exciting cause. Cranio-tabes might result from congenital syphilis, but hardly the formation of abnormal dentate and squamo-parietal sutures. Possibly, too, the syphilitic infection of the mother occurred subsequently to the birth of this child.

(*Exhibited May the 4th*, 1906.)

85. TUMOUR OF THE CEREBRUM; HYDROCEPHALUS.

By E. C. MACKAY.

(Introduced.)

V. R. B—, aged $5\frac{8}{12}$ths years, was admitted into the Royal Waterloo Hospital on January the 15th, 1906, under the care of Dr. Septimus Sunderland.

It was brought to hospital for "wasting." Patient is one of twins, the other died of "bronchitis" at eight months old. This child began to waste from the age of eight months. It was sent into the infirmary when three and a half and remained there two years.

Two or three years ago it had measles; there was no history of lues. The father has "phthisis" and has been in the infirmary for four years; the father's sister died of phthisis. The mother bore five children, then a miscarriage (twins), then no pregnancy for five years, subsequently twins, of whom this child is the survivor.

On admission.—The child was neglected, verminous, and little more than a "living skeleton," weighing only 15 lbs. 1 oz. The head was large, the fontanelle closed, the forehead prominent, and over the mid-frontal region a prominent plexus of distended veins was visible. There was a broad, sunken nose, and prominent wide mouth.

Eyes.—Some ciliary blepharitis; well-marked lateral nystagmus; pupillary reflexes natural; and vision seemed to be quite good. By ophthalmoscope: The optic papilla showed white atrophy and contracted vessels. There were small glistening patches of choroidal atrophy scattered widely over the fundus.

The chest and abdomen showed no evidence of disease beyond rickety changes and emaciation. The liver was not enlarged. There were no paralyses, and the child's movements were all quite purposeful and accurate. She had no symptoms except occasional vomiting, and that was never urgent. She was quite intelligent. *Course*: Progressive failure. At the end of February the hydrocephalus seemed to increase; there were occasional retractions of the head and increased nystagmus, with progressive failure of vision, so that in the end she could not see to play with her toys or take her food properly. She lay apathetic, with complete flexion of the lower limbs, and with complete incontinence.

Post-mortem examination.—Organs wasted but otherwise natural. No consolidation in lungs. Liver not enlarged, and showed no evidence of cirrhosis. Skull showed in left frontal region a depression of the outer table, which suggested an old-healed node. The edges were irregular and the base translucent. The inner table was not affected. There were two others on the inner surface which involved the inner table only. The brain showed the tumour and advanced hydrocephalus, as shown in the specimen produced. Microscopically the tumour was a glioma.

(*Exhibited May the 4th*, 1906.)

86. SKULL FROM A CASE OF CONGENITAL SYPHILIS AND HYDROCEPHALUS.*

By G. A. SUTHERLAND, M.D.

The skull shows well-marked cranio-tabes; also the results of intra-cranial drainage for hydrocephalus. The skull has contracted until the bones are over-riding.

(*Exhibited May the 4th*, 1906.)

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* For a history of this case *vide* Dr. G. A. Sutherland's remarks on a case of "Internal Drainage of the Ventricles in a Case of Hydrocephalus," by Mr. C. A. Morton (Bristol). 'Reports,' vol. iv, pp. 323–324. — Ed.

87. A SPECIMEN OF STOMACH SHOWING ULCERATION ROUND THE PYLORUS COVERED WITH DIPHTHERITIC MEMBRANE; ALSO MICROSCOPIC SECTION WITH DIPHTHERIA BACILLI.

By A. LEATHEM.

(Introduced.)

M. M. B—, aged 6 years and 9 months, was admitted into hospital for ulcerated throat. The child had been ill for three weeks with sore throat, cough, and vomiting, and died ten hours after his entry into the wards. A culture taken from the throat gave an almost pure growth of Klebs-Löffler bacillus. The specimens shown are: (1) larynx, with extensive membrane covering the tonsils, fauces, soft palate, and epiglottis; the membrane does not extend down the trachea, but there is a thick membrane covering the upper two inches of the œsophagus; (2) stomach showing ulceration round the pylorus, covered with diphtheritic membrane; (3) microscopic section of a piece of membrane from the pylorus, showing diphtheria bacilli. (*Exhibited May the 4th,* 1906.)

Discussion.

Dr. Theodore Fisher said it seemed to him rather a remarkable thing that ulceration of the stomach did not take place more often than it did, especially taking into consideration the large number of bacilli swallowed. It occurred to him whether the hydrochloric acid in the stomach was sufficient to kill the bacilli. Latterly he had not had any opportunity of examining such cases, but he thought it would be an interesting point to see how often diphtheritic bacilli were present in the stomach of children dying from diphtheria. If they can live there, they must be a fertile source of production of toxine.

Dr. Edmund Cautley asked Dr. Leathem whether he could say if this child was fed with a nasal tube, or whether there was any possibility of the mucous membrane of stomach being injured. Three years ago he exhibited before the Society a specimen of extensive diphtheritic ulceration of the stomach. Two small ulcers, one on each tonsil, were found post-mortem, but the case was not even suspected to be diphtheria during life. The question of how the infection spread was a very interesting one. Possibly the explanation was that the disease spread down the œsophagus by direct extension, while in other cases it was carried down into the stomach by means of a nasal tube, or through the swallowing of diphtheric bacilli. In this case the ulceration suggested to him that there had been some preliminary damage, possibly some necrosis, and secondary ulceration due to the bacillary infection.

Dr. A. Leathem (introduced), in reply, said the reason one did not meet with diphtheritic lesions in the stomach very frequently was probably due to the fact of hydrochloric acid being found in the stomach. This child had been ill for some three weeks, and he thought it extremely possible that the gastric juice was deficient in hydrochloric acid. The child had not been treated for diphtheria, and was practically in a state of collapse when brought to the hospital, and until then diphtheria had not been diagnosed.

88. THREE SPECIMENS OF CONGENITAL DEFORMITY OF THE HEART.

By J. G. EMANUEL, M.D.

In the first specimen (kindly lent by Dr. G. W. Powell) the aorta and the pulmonary artery arose from a single ventricle—probably the right ventricle. There was a functionless and rudimentary left ventricle which communicated with the aorta and also with the right ventricle. In addition to this the aorta was imperfectly divided externally from the pulmonary artery, and the pulmonary artery lay posteriorly to the aorta, as in the early stages of embryonic development. The inter-auricular septum was complete, the foramen ovale being closed. All the valves were normal. The specimen was from a male child, aged 20 months, that died of septic thrombosis of the right pulmonary artery, with multiple infarcts in the corresponding lung.

In the second specimen (kindly lent by Dr O. J. Kauffmann) the aorta arose from the right ventricle and the pulmonary artery from the left, and the foramen ovale was widely open. This specimen was from a "blue" boy, aged 11 years, with clubbed fingers and toes, who died of cerebral abscess the result of suppurative otitis media.

The third specimen was kindly lent by Dr. Stacey T. Wilson, and was taken from a "blue" boy, aged 7 years, with clubbed fingers and toes. The membranous part of the interventricular septum was absent, and the aorta arose as much from the right as from the left ventricle. There was a small but otherwise normal pulmonary artery with normal valves, which arose from a small conus arteriosus, which only communicated with the right ventricle by a narrow slit $\frac{1}{3}$rd inch wide. All the blood sent to the lungs during life had been driven by the right ventricle through this narrow aperture. (*Exhibited May the 4th*, 1906.)

Discussion.

Dr. Theodore Fisher thought one of these cases was a very interesting one because the boy lived to eleven years. There was a specimen, however, in the museum of the Bristol Royal Infirmary of a patient who died at the age of forty years.

Dr. C. W. Chapman said he had been wondering with regard to that double current (?) whether there was any difference in the density of the fluids. He could not see how they could otherwise have crossed. He also said he had been much puzzled with regard to the clubbing.

Dr. J. G. Emanuel (Birmingham), in replying, said the boy he referred to, who died, was eleven years old, and so far as the heart was concerned might have lived for many years had it not been for the middle-ear disease. One had to be very careful in taking into consideration any illness of the mother during pregnancy. There was no evidence of syphilis, and there was nothing in the history suggestive of it. He thought the cases were purely developmental in orgin, and that the question of syphilis had no part in their production at all.

89. SPECIMEN OF CONGENITAL MORBUS CORDIS.

By GEORGE CARPENTER, M.D.

THE specimen shows two cusps to the pulmonary valve There is an ill-marked division in one of them. Also a patent septum ventriculorum about $\frac{1}{4}$ inch in diameter. The aorta is of normal size and arises naturally. The pulmonary artery is dilated. The foramen ovale is closed, and the ductus arteriosus is obliterated. The right ventricle is hypertrophied, the left normal.

The heart was removed from Mary A—, aged 2 years 3 months, on March the 20th, 1906. She was born a "blue baby," never seemed to thrive, and always had a bad cough in the winter. A fairly well nourished child. Bulging of the præcordia, marked pulsation in the epigastrium, apex beat visible in the fourth interspace in the nipple line. Marked systolic thrill all over cardiac region. The cardiac dulness extended only slightly to the right of the sternum. A loud, rough, systolic bruit was audible all over the chest, the point of maximum intensity being over the pulmonary area; it was conducted down the left side of the sternum, and it was not audible in the big vessels of the neck. Spleen enlarged. No clubbing. Second pulmonary sound accentuated. Death from broncho-pneumonia.

(*Exhibited May the 4th,* 1906.)

90. A SPECIMEN OF PRIMARY SARCOMA OF THE LUNG.

By F. LANGMEAD, M.D.

The specimen was removed from the body of a girl, aged 5 years. She was admitted into the Great Ormond Street Hospital for Sick Children under Dr. Lees, on account of cough, wasting, and pain in the chest and left arm of five months' duration. The signs during life were those of pleural effusion, and this was the diagnosis with which she was admitted. The whole of the front of the left side of the chest, and the back on the same side down to one finger's breadth below the angle of the scapula, were very dull and resistant. Breath sounds were almost absent in front, and inaudible behind over the dull area. Vocal fremitus and resonance were both absent. The heart was displaced to the right, its right border reaching for two inches outside the right sternal margin, whilst the impulse was strongest in the epigastrium. The spleen was easily felt two fingers' breadth below the costal margin. On the left second rib, just in front of the anterior axillary line, was a swelling as large as a pigeon's egg. This was not tender, and not adherent to the skin, it felt somewhat hard, and did not fluctuate. An exploratory needle was inserted into the back, into the mid-axilla, and into the rib-swelling, but blood only was withdrawn. Four days later an incision was made over the rib-swelling, and it was found to be new growth which had spread from within the chest and had become adherent to the rib. A slice was removed for microscopical examination, and proved the tumour to be a round-celled sarcoma. The child died after six weeks, during which time she became progressively more dyspnœic and cyanosed, and wasted rapidly. The signs in the chest did not alter. There was never any expectoration.

Post-mortem examination.—The left side of the chest was filled by a large mass of new growth, which had spread across the mid-line behind the sternum. It was evidently situated within the lung, and was strictly limited by the visceral pleura, except in the upper and outer part of the thorax. Here it had spread through the intercostal spaces, causing destruction of the ribs and projected externally, forming the swelling previously noticed. The tumour was limited to the upper lobe, which had been completely transformed except for a narrow strip on its inner aspect. The left lower lobe was in a state of collapse and fibrosis. The mass was muddy-grey in colour, and everywhere soft. In many parts the consistence was that of thin mortar, especially so in the middle of the growth. In a few places hæmorrhage had occurred. Stretched over it was the vascular visceral pleura. The pleural cavity contained no new growth, except where it had come to the surface. No growth was found elsewhere..

Primary sarcoma of the lung is a very rare disease. Since 1860 there has been but one previous post-mortem on such a case at the Great Ormond Street Hospital for Sick Children, which is recorded in the 'Transactions of the Pathological Society,' 1903. Dr. Samuel West, writing in 1902, could only find five examples, none of which were in children. Drs. Rolleston and Trevor record a case in 1903 in a girl aged 13 years, and add two more in adults from the records of St. George's Hospital. Another case is recorded by Dr. Box, in a girl aged 5 years.

It is noteworthy that in all four cases which occurred in children a needle was inserted more than once, under the conviction that fluid was present.

(*Exhibited May the 4th*, 1906.)

Discussion.

Dr. C. W. Chapman asked whether in the early stages there was any bronchial breathing or any particular patches of bronchial breathing.

Dr. F. Langmead, in reply, said that the girl had not been seen medically until there had been pain and cough for five months, so that the time for early signs was over. When seen, the signs were absolutely those of fluid.

91. A SPECIMEN OF MENINGEAL HÆMORRHAGE IN WHOOPING COUGH.

By F. LANGMEAD, M.D.

The specimen was removed from a girl aged 2 years. She was admitted into the Great Ormond Street Hospital for Sick Children under Dr. Colman on account of whooping cough of fourteen days' duration, complicated by bronchopneumonia. About fourteen days after admission twitching of the left arm and hand and left eyelid was noticed, which recurred on the same day. Two days later there was conjugate deviation of the eyes to the right. The next day there was frequent twitching of the left arm and face, and occasionally of the left leg, especially when she was disturbed for feeding purposes. The spasms were at the rate of one per second. Later in the day they occurred on the right side as well. This condition remained until death, at 6 a.m. next morning. The coughing had not been especially severe before the onset of the nervous symptoms.

Post-mortem examination.—On the vertex of the brain was a large area of meningeal hæmorrhage, forming a dark red coat over the whole of the vertex on the right side and the anterior two thirds of the vertex on the left. The base was almost free from hæmorrhage. On the lateral aspects of the hemispheres it was not, as on the vertex, in a continuous sheet, but punctate in character, showing that it was due, not to rupture of a vessel, but to general leakage from the cortical veins. The veins were intensely engorged and filled with red and mixed clot. The sinuses were also distended with clot. (*Exhibited May the 4th,* 1906.)

Discussion.

Dr. Theodore Fisher said it seemed to him that the hæmorrhage was very possibly secondary to thrombosis of the cerebral veins. There certainly was thrombosis of the veins. He had had four similar cases under his notice, and only in one of them was there any marked cough.

Dr. F. Langmead, in reply, said he was very glad to hear of previous examples where the meningeal hæmorrhage was like it was in this case and yet no whooping cough was present. He could not help feeling that it was an extremely rare complication of whooping cough, because out of some 150 post-mortems at Great Ormond Street on this disease he had not found another example. It seemed possible that the thrombosis was primary, but he could not help thinking that the tremendous strain of the paroxysmal cough must have had some influence on the affected blood-vessels, and so determined the bleeding.

92. TUBERCULOUS OSTEITIS OF THE SKULL AND LOWER JAW.

By F. LANGMEAD, M.D.

The specimens were removed from a girl, aged 1½ years, who was under the care of Drs. Garrod and Batten at Great Ormond Street Children's Hospital. She was admitted on February the 25th, 1905, because of the presence of multiple rounded swellings on the head. The illness was stated to have commenced five months previously by a swelling in the lower jaw. The head swellings had been noticed for one month. The appearance of the head was very like that due to multiple sarcomata. Over the vertex, but especially in the frontal and left parietal regions, were soft, rounded, semi-fluctuant tumours, the longest having diameters of from 1 to 1½ inches. They gave an impression of being situated beneath the periosteum, and not involving the scalp, which was bulged, but otherwise normal. On the left arm were two skin tuberculides. The right side of the lower jaw was enlarged by a localised swelling. A week later, to settle the diagnosis, a needle was inserted into one of the more fluctuant tumours, and a film stained by the curdy material removed. In this tubercle bacilli were found. From this time onwards many fresh foci of tuberculosis developed, including one in the left malar bone, one in the left arm, and one in the left calf. Some rapidly softened, and required incision and curetting. Two months later the swellings on the head had become much reduced, and many elsewhere had gone altogether. She was discharged, but returned after two months' absence with fresh cranial swellings and dactylitis. While in the hospital she developed meningitis, and died, ten months after the first symptoms of her illness.

Post-mortem examination.—Skull: In the bone were several areas of tuberculous osteitis. The larger of these were situated in the left parietal bone near its anterior inferior angle. In this region were four irregularly circular areas, each of about the circumference of a shilling, in which the bone, with the exception of the inner table, was replaced by caseous material (in the specimen the periosteum had been stripped off). These foci were slightly elevated above the surface, and surrounded by slightly undermined edges of porous bone. Elsewhere were several similar but smaller areas in various stages. In some there was destruction of the outer table, in others the condition was still limited to the diploe, in which apparently the process started in all. The inner table had in no instance been destroyed.

Lower jaw: In the body of the lower jaw on each side were similar foci of caseous osteitis. That on the right side had communicated with the mouth, and had consequently been secondarily infected and undergone putrefactive changes; that on the left was about as large as a pigeon's egg, and was still confined within the bone, the outer table being apparently normal.

Similar changes were found in the malar bones, whilst there was also tuberculosis of the meninges and bronchial glands.

(*Exhibited May the 4th*, 1906.)

93. SECTIONS OF TUBERCULOUS ULCERATION OF THE STOMACH.

By F. LANGMEAD, M.D.

The macroscopical specimens have been shown at a previous meeting (February the 16th, 1906, *vide* pp. 184–186).

Section 1 was taken from a case of general miliary tuberculosis in a girl aged 8 months. In the stomach were eight yellow tubercles situated on arterial twigs. The section shows a tiny ulceration of the mucosa, and at its base a focus of round-celled exudation, in which caseation has commenced. Many tubercle bacilli can be seen in the base of the ulcer.

Section 2 was taken from a boy, aged 1 year and 11 months, who died of general tuberculosis. The ulcer was situated on the anterior wall of the stomach, and was about 9 mm. in diameter. The section shows the typical undermining of tuberculous ulceration. In this specimen, too, caseation has commenced. Lying among the necrotic cells at the base of the ulcer are several tubercle bacilli. There are no giant-cell systems in either section.

(*Exhibited May the 4th,* 1906.)

94. NÆVI CYSTEPITHELIOMATOSI DISSEMINATI (LYMPHANGIOMA TUBEROSUM MULTIPLEX).

By GEORGE PERNET.

This is a very rare disease, consisting of numerous small tumours scattered about the upper part of the front of the chest mainly, but occurring also on the back, face, etc. The specimen shown was taken from a female patient, aged 25 years, in whom the growths are said to have first appeared at sixteen. The histological appearances were, briefly, small cysts with colloid contents and strands and aggregations of cells, which the exhibitor considered were of epithelial origin. There were many names for the condition, according to the view as to origin held by individual observers—lymphatic, vascular, sudoral, endothelial, and epithelial. The exhibitor, as a result of the sections of the case he had examined, favoured the epithelial view, and considered that the term "nævi cystepitheliomatosi disseminati" best described it, using the word nævus in its widest sense, viz. in that of a congenital cutaneous blemish or growth. If this view turned out to be correct, as it appeared to be from the evidence brought forward by others, as well as by the exhibitor, the importance of it from the Cohnheim theory was obvious.

(*Exhibited May the 4th,* 1906.)

95. RACIAL PIGMENTATION ON THE BUTTOCKS.

By GEORGE PERNET.

A coloured drawing and photograph of racial pigmentation on the buttocks of a Siamese baby, received from Dr. Gimlette, of Kelantan, Siam, were shown by Mr. George Pernet.

(*Exhibited May the 4th,* 1906.)

96. SPECIMEN OF TUBERCULOSIS OF AN INGUINAL HERNIAL SAC.

By H. S. CLOGG, M.S.

Removed from H. C—, aged 5 years. Right inguinal hernia had been noticed for twelve months. At operation the sac was an incomplete one, not communicating with the tunica vaginalis. It was thickened and studded on its interior with miliary tubercles. An ordinary radical cure was performed. On examination of the abdomen under anæsthesia there was some indefinite "lump" palpable in the hypogastric region. There was no free fluid present. The testicle was normal. The child had never had any symptoms of tuberculous peritonitis.

The microscopical section shows the usual giant-cell systems of tuberculous disease.

(*Exhibited May the 4th*, 1906.)

97. SPECIMEN OF FIBRO-SARCOMA OF THE PAROTID GLAND.

By H. S. CLOGG, M.S.

Removed from E. R—, aged 11 years. Tumour in the right parotid region, noticed for three months, of slow growth. The tumour was imbedded in the substance of the parotid gland, and was easily enucleated.

Microscopically the appearance is a fibro-sarcoma.

(*Exhibited May the 4th*, 1906.)

98. SPECIMEN OF ANGEIOMA OF THE PAROTID GLAND (?) ANGEIO-SARCOMA.

By H. S. CLOGG, M.S.

Removed from A. P—, aged 7 months. Tumour in right side of face had been noticed for four months; rapid growth of late. Tumour measured about $\frac{3}{4}$ths inch in diameter. It was imbedded in the substance of the parotid gland. It was dissected out from this with some difficulty. The hæmorrhage was free; on the deep surface the facial nerve was exposed for some distance, but not wounded. The tumour had no definite capsule, and could not be enucleated.

The tumour is made up of vascular tissue with a very cellular stroma. In places the endothelial cells lining the vessels are proliferating. Areas of gland tissue are seen in the section. The rapid growth, non-encapsulation, infiltration of the gland, and the cellular stroma seem to point to the tumour being a sarcoma, possibly of endothelial origin. (*Exhibited May the 4th*, 1906).

99. TUBERCULOUS KIDNEY REMOVED BY OPERATION FROM A GIRL AGED TEN YEARS.

By GEORGE CARPENTER, M.D.,

AND

EWEN STABB.

(Introduced.)

The kidney presents the typical appearance of tuberculous disease. At the lower end is a cavity which still contains white granular material—caseous material mixed with calcareous salts. The remainder of the cavities show typical thick walls lined with caseous material. The specimen also has attached a large portion of the ureter cut away as low down as possible at the time of operation. It is much thickened and hypertrophied, and shows typical tubercular lesions.

The child, a girl aged 10 years, had suffered from "pains in the stomach, and, at times, passing blood in the urine," the former of a year's duration, the latter for a month. At first the "pains" were slight, but they became worse. She was admitted into hospital on February the 22nd, 1906. During the month before she was admitted into hospital she had twice passed blood, preceded by colicky pain. She had great frequency of micturition and nocturnal enuresis, and pains in the head in the occipital region, especially at night. She also complained of pain on making water. She was rapidly wasting, but took her food well. There were five in the family, all alive and well. No tuberculous family history.

On admission on February the 22nd she was found to be a pale and emaciated child. Abdomen easily palpable, nothing to be felt. Heart and lungs healthy. During the first night of her stay in hospital she passed urine twelve times. The urine on the second night was bright red, contained blood, and a considerable amount of pus in acid urine. This appearance of blood followed a severe paroxysm of pain in the left groin, extending down to the left thigh, which lasted in paroxysms for two hours, and was followed by hæmaturia, which ceased eight hours later. On this date, after a thorough evacuation of the bowels, both kidneys were palpable, the left one appeared to be enlarged, and pain was evinced on palpation. On the 24th there was hæmaturia in relation to paroxysmal pain in the left kidney region, also on the 25th. Tubercle bacilli were examined for on the 24th, but none were found, but on the 25th they were detected in abundance in clumps—the gangrene bacillus was excluded—and blood and pus were seen by the microscope. On the 9th, under an anæsthetic, the bladder was examined by Kelly's cystoscope. The mucous membrane was found to be much thickened and granular. Owing to the state of the mucous membrane and the bloody urine it was very difficult to locate the orifices of the ureters. A catheter was passed into the right ureter, and a specimen was drawn off. A catheter was passed into the left, but the urine would not flow. The urine from the right kidney was examined by the pathologist, and he reported that "the deposit consisted of multinuclear and large mononuclear cells. The mononuclear cells were apparently cells from the pelvis of the kidney. No micro-organisms were seen." A further examination was made a week later, when catheters were passed into both ureters; clear urine was drawn from the right kidney, from which it flowed freely. From the left a large amount of thick urine escaped, accelerated by pressure in the region of the left kidney, and large numbers of tubercle bacilli were found in this. Tubercles were seen about the orifice of the left ureter. Between the 9th and this latter date paroxysms of pain had continued on and off, and the urine had at times contained blood in relation with these paroxysms. There was always a large deposit of pus, the reaction was acid, and there was no smell. During the whole period during which she had been under observation micturition had been exceedingly frequent and in small quantities, and rest in bed had not improved the condition. On March the 20th the left kidney was excised by lumbar nephrectomy, and the ureter was dissected out to within an inch and a half of the bladder. The wound healed by first intention. On April the 20th there was still frequency of micturition, pus, and albumin in the urine; tubercle bacilli also present. Her general health was improving, and she was putting on flesh. The radiographer reported on March the 19th, the day before the operation, that there was an opaque shadow in the region of the left kidney, which the radiographer thought to be stone.

(*Exhibited May the 4th,* 1906.)

Postscript, October, 1906.—On May the 16th the bladder was examined by Kelly's speculum, which revealed extensive ulceration. A suprapubic cystotomy was performed, and the interior of the bladder appeared like a granulating cavity, there being no healthy mucous membrane to be seen. After the use of the sharp spoon in several places the whole surface was well rubbed with nitrate of silver solution, 20 grains to the ounce, and the bladder was drained above the pubis. Suprapubic drainage was discontinued on July the 27th, and the opening was allowed to close. On October the 26th she was sent to a convalescent home. Her general condition had much improved. The urine now passed naturally, but still contained a variable amount of albumin and pus.

100. BRAIN FROM A CHILD AGED SIXTEEN MONTHS THE SUBJECT OF HEAD-BANGING.

By GEORGE CARPENTER, M.D.

On removing the dura mater the appearance of the brain was peculiar, somewhat resembling that of a general paralytic. Clear, œdematous-looking fluid lay between the layers of the pia arachnoid. The arachnoid itself was generally slightly more opaque, and here and there were definitely opaque patches. The noteworthy feature was the increase in the number of medium-sized blood-vessels on this membrane. This was especially noticeable when the brain had been removed. Meshes of vessels were then seen running upwards in the Sylvian fissures in the thickened membrane, which floated on fluid between it and the pia mater. At the commencement of the fissure the membranes were much thickened and opaque, they were also thickened on the under surface of the pons. The ventricles were not dilated, but there was thickening in places of the ependyma, the underlying vessels sometimes being completely hidden. The convolutions over the frontal region were smaller than normal. The brain was natural in size, no evidence of ante-mortem clot in the superior longitudinal sinus. The lungs were adherent to the chest by loose adhesions on both sides; no evidence of tuberculosis or tuberculous lesions of the bronchial glands; on section pus exuded from the bronchioles and patches of broncho-pneumonia were seen. No post-mortem evidence of syphilis.

The brain was removed from Sydney B—, aged 16 months, on November the 28th, 1905. He was admitted for "banging and smashing his head about" on November the 23rd, 1905. At five weeks old he had "screaming fits." At four months old "he again began to bang his head about, and did not seem to see." At 4½ months he had a "rash," and a discharge from the nose. Following that he had "boils all over the body and arms." On admission he was anæmic, he had a cough and snuffles, and had cut four teeth. The ribs were beaded, there was slight enlargement of the epiphyses, and the anterior fontanelle was not closed. The physical signs were those of broncho-pneumonia. In regard to the family history there were nine children, eight of them being alive; the death was due to one of twins dying at the birth. In addition to the nine children there were three miscarriages, two before the birth of the patient, and one born dead. Breast-fed for two months, subsequently on cow's milk and patent foods. The temperature for the five days he was an inmate of the hospital was normal, and his pulse rate varied between 108 and 140, and his respiration rate between 24 and 40.

(*Exhibited May the 4th,* 1906.)

101. LUNG WITH MULTIPLE ABSCESSES FROM A CHILD AGED THREE YEARS WITH PYO-PNEUMOTHORAX.

By GEORGE CARPENTER, M.D.

THE right lung was collapsed and covered with a thick purulent exudation. A band of adhesion between the middle and upper lobes, and the chest wall held the lung in front against the parietes, which divided the pleural cavity into two parts. Pus on the inner side of the lung between it and the mediastinum had not been able to flow freely into the rest of the pleural cavity, and several ounces remained there, whereas the part that had been opened at the operation had drained freely. Below, the lung was firmly adherent to the diaphragm. At the anterior extremity of the lower lobe three apertures were visible, all of which opened into the abscess cavity. The whole of the lower lobe appeared to be composed of small abscess cavities, apparently broken down broncho-pneumonia. The upper lobe was almost free from disease, and the left lung only contained a few patches of broncho-pneumonia. Cover-slip preparations from the pus showed a very large coccus, which stained very deeply with methylene blue. It occurred chiefly in small groups of four or five to six, and chiefly as a diplococcus.

The heart was much displaced to the left, the right border of the right auricle being about the centre of the sternum.

The child, a boy aged 3 years, was admitted into the North-Eastern Hospital for drowsiness with the following history. On November the 13th anorexia, on the 14th he vomited twice, was drowsy, slept most of the day, and had a slight cough—thought to be bilious. On the 16th he was much worse, was very feverish, had a sore throat and a catch in the breath, and loose green stools. He was admitted on November the 17th, 1905. On examination he was a wasted boy with sunken eyes, a clean tongue, and easy, though quickened, breathing, and he breathed without distress even when lying down. When disturbed his respiration became grunting, and he resisted being interfered with. His chest was resonant back and front when examined in bed in the ordinary way. On the right side in front above the nipple the breathing was vesicular, with just a suspicion of an amphoric character, which latter became more marked as the nipple was approached. Below the nipple the breathing was decidedly amphoric, and became increasingly so down to the liver, together with metallic tinkling which was superadded. In front a *bruit d'airain* was audible in the lower two thirds of the chest, and in the axilla all over the chest. Behind, at the right root, the breathing was bronchial in character, but elsewhere it was vesicular and associated with fine crackling. When laryngeal grunting occurred the grunt terminated in a metallic tinkle, which was audible in the neighbourhood of the angle of the scapula. When the child was shaken splashing was readily heard, and also a tinkling sound. When he was bent forwards the front of the chest became dull to percussion, and amphoric breathing disappeared. When he was tilted backwards the back became dull. The heart was displaced to the left, the apex beat being in the fifth interspace half an inch outside the nipple line. The cardiac dulness commenced one and half fingers' breadth to the left of the *left* sternal margin. It was absolute at the fourth left costal cartilage, and considerably modified at the third left costal cartilage. Viewed with the X rays the front of the chest was transparent except over a triangular strip, with its base downwards reaching from the third rib to the sternum and liver, which was opaque to the rays, but not so opaque as the heart. Looked at from behind, the affected side was transparent, and it became increasingly so, and more so than the normal in the course of a few hours. Moreover it was much narrower, less by one third than on the normal side. There were no signs of a lung behind, and it was thought on that account that the opaque triangular patch aforesaid was lung, but where this was situated was not quite obvious. Another idea was that the patch was a piece of pneumonic lung, the rest being transparent. The extreme displacement of the heart was considered to be due to extreme pressure of air in the pleural cavity. The diaphragm of the affected side was distinctly concave above and barely moved with respiration, whereas the other side moved very freely, carrying the stomach with it on its excursions. The chest was opened and six ounces of pus were evacuated, but he died the same night.

Remarks.—Pneumothorax is an uncommon disease in childhood, but I have seen two other cases since I have been in practice, both of which were mentioned in the observations I made on "Pleural Effusions, Serous and Purulent," at the December Meeting, 1905, of this Society, p. 115. In a boy aged 3 years, with empyema, there was free air in the pleural cavity. The right lung was collapsed, the pleural cavity being divided into two by dense adhesions provided with openings. A small abscess cavity in the lower lobe of the lung filled with caseous material communicated with a bronchus, but not with the pleura.

In a boy aged 14 months, with empyema, the left lung was collapsed. In the left lower lobe of the lung in which there was a small localised abscess, was an orifice connected with a superficial cavity and indirectly with the bronchi. On the under surface of the lung were three small apertures communicating with a network of ragged cavities partially separated by bridges of shreddy tissue, and every here and there were portions of solid lung-tissue becoming gangrenous. (*Exhibited May the 4th*, 1906.)

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102. INTERSTITIAL NEPHRITIS AND CIRRHOSIS OF THE SUPRARENAL CAPSULES IN AN INFANT FIVE WEEKS OLD.*

By GEORGE CARPENTER, M.D.

Although physiological albuminuria is common enough in the newly-born, yet albuminuria secondary to obvious renal disease appears to be quite the exception, and it is still held by many that interstitial nephritis in infants is one of the rarest of diseases. It may be, however, that kidney troubles are not so rare in them as we have hitherto supposed to be the case, and that more careful observations would increase our knowledge, and, at the same time, alter our present views. There is no doubt that the infantile kidney may be seriously diseased and yet not show any pathological changes whatever to the naked eye, and would therefore at the autopsy be deemed normal and recorded as such. It follows, therefore, that these organs cannot be pronounced sound on a mere ocular inspection at the post-mortem examination, and that it will be necessary in the future to revise our methods of research, and rely more upon the microscope than has hitherto been the custom. The following case is not only an example of an apparently uncommon condition of the kidneys in a newly-born infant, and probably of intra-uterine origin, but it illustrates changes in the supra-renal capsules which I have not met with before, and which I cannot recall as having read of in the literature.

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* 'Lancet,' November the 3rd, 1906.

Harold B—, aged 5 weeks, was "healthy and well-nourished" at birth. About the tenth day he "began to pine." He was breast-fed and an eight months' child. A brother of the patient died from "eczema." He was admitted into the North-Eastern Hospital for Children on January the 18th, 1906, with "diarrhœa and vomiting." The stools were green and contained curds, and in one there was a small amount of blood. Previous to admission there was a history of passing blood by the bowel and also blood from the penis.

On examination.—His face was puffy and his back very œdematous and pitted on pressure. The hands and feet pitted, the latter especially, and the upper extremities were œdematous as far as the shoulders, and the lower extremities as far as the trunk. The abdomen was very distended with wind, there was some free fluid there, and the skin was pale.

Heart.—A systolic bruit was detected and was heard best at the left base, not especially loud there, but it was conducted all over the front of the chest, though inaudible over the back when the child was crying. The pulse could not be felt at the wrist. The lungs were normal. Urine albuminous. The testicles were neither large nor hard.

He vomited seven times on the date of admission into the hospital, and once or twice daily the preceding days. The bowels were opened once or twice daily.

He died on January the 23rd, 1906, and an autopsy was made on the same date.

Lungs.—A state of mixed broncho-pneumonia and collapse was found down the back of the right lung.

Heart.—In spite of the abdominal distension, the apex of the heart was in the fifth interspace.

Abdomen.—On opening the abdomen a quantity of clear fluid amounting to 4½ oz. flowed away. The peritoneum was shiny, and there was nothing to indicate acute peritonitis, but in parts, however, there were appearances that suggested chronic peritonitis. In places there appeared to be some alteration over the coils of the small intestine where the peritoneum seemed thickened ; there was a network of vessels surrounding the coils, but the network was coarse, not fine, such as is seen in acute inflammatory conditions, while the peritoneum over the intestines had not the opacity seen in chronic peritonitis. The mesentery in the neighbourhood of the congested area was pigmented. Many of the glands in the vicinity were also pigmented. There were many adhesions between the liver and the abdominal wall.

Liver.—Anæmic patches were seen, but the organ appeared normal to the naked eye.

Spleen.—This was enlarged to twice the normal size.

Kidneys.—These were normal *to the naked eye*, but microscopically the structural alterations were as follows: the capsule was thickened, interstitial changes showing chronic inflammation were readily seen, the glomeruli were atrophied, and the epithelium of the tubes was shed and necrotic, the changes being most marked in the cortex, and here some thickening of the small arteries was detected.

Suprarenal capsules.—Both these organs were adherent to the kidneys, and they were enlarged, and of opaque, yellowish-white colour. Sections of the right showed what appeared to be an old hæmorrhage. Microscopically chronic inflammatory changes were found.

Liver.—The liver presented some peculiarities microscopically. There were large numbers of lymphocytes and other leucocytes in the veins, not in the arteries, and also in the capillaries.

Pancreas.—Normal.

Brain.—Not examined.

Remarks.—There were neither history nor stigmata of syphilis in this infant, but the lesions discovered postmortem were doubtless of a syphilitic nature, and most probably of extra-uterine origin.

Cases of renal disease in infants have been reported to the Society by Dr. Henry Ashby,[*] by myself,[†] and also by Dr. G. A. Sutherland and Mr. J. W. Thomson Walker,[‡] in this case of a girl of sixteen months old.

Dr. Ashby's case was four weeks old; it, like my patient, was anasarcous. On section the cut surface of the kidney was pale and fatty-looking, and there was very little difference between the cortex and medulla. The microscopical examination showed "choked" kidneys, with extensive epithelial and fibroid changes, and the small arteries appeared thickened. There was no evidence of syphilis, but it appeared to me at the time the specimen was shown that it might, notwithstanding the lack of positive evidence, be of syphilitic origin.

The case I reported was dropsical, five months old, and syphilitic. The urine was albuminous and contained hyaline, epithelial, and granular casts. Coarse alterations were not obvious in the kidneys. Microscopically there were catarrhal changes, glomerular over-nucleation, and thickening of the small arteries in the neighbourhood of the glomeruli. I also, when recording this case, narrated the case of another syphilitic and dropsical infant, aged 5 weeks, I had seen some years before, and published in my book on "Syphilis in Children," who had glomerulitis, and interstitial and catarrhal nephritis, with hyaline and epithelial and a few blood casts. The kidneys in this patient also *appeared healthy* macroscopically, and this fact was then commented upon.

Dr. Sutherland and Mr. J. W. Thomson Walker's case was a syphilitic child sixteen months old. On one occasion its urine was drawn off by catheter and contained a small amount of albumin, but attention was not called by the symptoms to the kidneys, which post mortem showed marked interstitial changes widely separating the tubules. Many tubules were filled with casts of homogeneous material or masses of round cells. The majority of the glomeruli displayed no change; some showed a slight increase in their nuclei.

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[*] "A Case of Nephritis in a Newly-born Infant," the 'Reports of the Society for the Study of Disease in Children,' vol. i, pp. 129–132.

[†] "A Case of Syphilitic Nephritis in an Infant aged Five Months," the 'Reports of the Society for the Study of Disease in Children,' vol. iii, pp. 286–295.

[‡] "A Case of Syphilitic Endarteritis and Nephritis in an Infant," the 'Reports of the Society for the Study of Disease in Children,' vol. iii, pp. 134–146.

They also on that occasion narrated another case of a syphilitic girl of eight months, whose kidneys showed similar interstitial changes to the above, but the glomeruli and tubules were normal. In the discussion that followed the writer also gave the history of a syphilitic infant of six months, who had albuminuria and anasarca of six weeks' duration. It was lost sight of, but probably died.

Although cases of infantile renal disease have been previously brought to the notice of the Society, this is the first instance of cirrhosis of the suprarenal capsules that has been recorded. The importance of not passing the kidneys as healthy on the evidence of a macroscopical examination alone is once more exemplified.

(*Exhibited May the 4th,* 1906.)

103. THE WIGHTMAN LECTURE: ON CHRONIC APPENDICITIS AND THE EARLY DIAGNOSIS AND TREATMENT OF ACUTE APPENDICITIS IN CHILDREN.

By A. BROCA, M.D.

(Introduced).

Mr. Chairman and Gentlemen,—I could scarcely resist a feeling of pride in speaking before you on this important occasion, were I not called back to modesty by an exact appreciation of the position. For, if you summoned me to deliver this lecture, by which Mr. Wightman desires to perpetuate the memory of a son who too soon departed this life, I am aware that I am indebted for this testimony of esteem to another which my Parisian colleagues, too indulgent also, had favoured me with last year, in calling me to the presidency of our Pediatric Society in Paris.

As their President, I had the pleasure to receive some of you (not numerous enough—allow me this reproach) when you came to Paris and visited our hospitals and great medical establishments. To my French colleagues, through me, is therefore addressed your courteous invitation and appreciation.

"Vous aviez d'ailleurs tiré les premiers, Messieurs les Anglais," by prompting us a few months before to proclaim medically our recent "entente cordiale." With a great feeling of pleasure I personally saw the end of our petty squabbles, which ought by mutual consent, and a long time ago, to have been immolated upon the altar of our common deity—freedom. I am glad to have the opportunity of giving my opinion here, for at that time I was prevented from coming to London, and I can assure you that it was not from any lack of sympathy with the welcome movement.

Now that I have apologised for my unworthiness, I must confess that I answered your invitation with the greatest pleasure; but I should hardly dare to speak before you on such a common topic as the one I have selected had not the first idea of lecturing on this subject come from your Chairman of Council—Mr. Clement Lucas—who had been struck, when he visited my hospital wards, by the great number of children operated on by me for appendicitis. He thought that I might interest you in relating what I have observed these fifteen past years in children, at an age when his own experience seems to suggest that this complaint is more frequent in Paris than in London.

Some authors think that this frequency in childhood does not depend upon a true predisposition: the children being the most numerous amongst the population, the same proportion of cases for a disease whose real frequency is not modified by age must give a larger number in the young. But that alone is not sufficient to explain such a predominant number of cases as we meet with in Paris, where, as soon as appendicitis began to be narrowly studied, important statistics appeared from our two children's hospitals, observed by our regretted Dr. Brun, by Jalaguier, and by myself. I will recall the fact that in 1896 one of my pupils, Miss Gordon, made her inaugural dissertation from my first seventy-nine observations on appendicitis in children, and since then I have operated more and more on patients under fifteen years of age suffering from appendicitis, partly because we have learned to diagnose some cases formerly unknown, and also, it seems to me, because we have had periods of increase, due to the frequent return of influenza to which we have unfortunately been accustomed since 1890.

But if, as I believe from what Mr. Clement Lucas wrote me, the frequency in childhood is greater in Paris than in London, I do not think that influenza is the true cause of that difference, for influenza has now invaded all countries. If there is a particular cause, my opinion is that it must be sought in the food.

In spite of some opponents, every day lessening, it is forcibly asserted nowadays that appendicitis is but the consequence, the localisation, of an entero-colitis previously more extended. This localisation gets its surgical importance on account of the special anatomical connections between the peritoneum and the appendix, predisposed by its structure to lymphangitic inflammation, even to perforation. But it also seems that all cases of enteritis have not the same tendency to create appendicitis.

Every one of us knows well how frequent is gastro-enteritis among babies not suckled, and who, suffering from diarrhœa or constipation, insufficiently fed or overfed, end, according to the cases, in athrepsia, summer diarrhœa or rickets. It is a fact, nevertheless, that appendicitis is almost never seen in new-born babies. The youngest on whom I have operated was 1 year and 8 months old, and such an age is very exceptional.

One of the causes of this apparent rarity in infancy is, no doubt, the serious clinical difficulties we have to deal with. We are bound, with very young children, to practise an examination almost veterinary, on patients unable to give us any information, unless by screams when we rouse pain by palpation. So the chronic forms, of which I shall speak to you by-and-bye, escape us, and with them no doubt a great number of acute ones. But the fact remains that our medical colleagues, in their post-mortem examinations of nurslings dead from gastro-enteritis, never mention peritonitis of appendicular origin in children under two years of age; they do not even describe the appendix and peri-appendix lesions whose importance we know well in older patients.

This is partly owing to the fact that appendicitis takes a certain time for its formation and for its surgical individuality after the beginning of the causal enteritis; and I think that a few children attacked with appendicitis suffer from food badly digested in infancy. However, I do not believe this to be a rule, and by the observations made at every age and in every country, it appears that the general medical opinion is that an excess of flesh diet may be the origin of enteritis predisposing to appendicitis.

But if you English people, men of the north, eat a great quantity of meat when grown up, you are in the habit of giving but little of it to your children during their first years of life—sometimes, perhaps, not even enough, if I may judge by what our French mothers call bringing up children "à l'Anglaise."

Among the predisposing forms of enteritis is one whose frequency has increased, during the last years, in childhood and in adults; it is muco-membranous enteritis. I insist upon it, for some persons have found a certain antagonism between that illness and appendicitis; but I think their view is precisely the opposite to the reality.

This point presents a double interest: (1) for the diagnosis; (2) for the treatment.

It is important from the point of view of diagnosis, for I have seen children die, and others almost dead, from repeated and unrecognised attacks of appendicitis, because appendicitis in their case was associated with evident attacks of muco-membranous enteritis; and those children's physicians, although learned and most distinguished doctors, have sorrowfully learnt how wrong was the mischievous doctrine of antagonism.

It is quite as important for the treatment, for it often happens that we are obliged to submit our patients to medical and dietetic treatment for enteritis after their appendices have been removed. I know well that in every case of muco-membranous entero-colitis there exists an important nervous element. Some of them are positively reflex and disappear with surprising rapidity after the surgical cure of abdominal disease—renal, utero-ovarian, etc. This may happen after the excision of a chronically irritated appendix; chiefly we have often observed to disappear, after its removal, old and intense constipation, due to intestinal atony, from which many patients suffer before the operation. We do not obtain the same results in muco-membranous diarrhœa, and I am compelled to record the cases of patients who suffered from more or less serious relapses after the operation for appendicitis.

By clinical observation it is not always possible to determine with what certainty an operation would cure intestinal disorders; but if any time before the operation these symptoms are intense and prolonged, it will be prudent after the surgical procedure to recommend for some months a careful medical treatment.

I said that, when enteritis exists, appendicitis takes some time to develop; and I add that most often this process is a chronic one. Formerly we did not think so; for, struck by the acuteness of the attack, we have not always inquired into the antecedents of our patients for the indications of a former and chronic lesion; but we were not long before realising that those symptoms existed, and almost all of us now regard appendicitis as a chronic alteration, aggravated by acute episodes. This notion has become common nowadays, but it was less so seven years ago, when I drew attention to it in a monograph on appendicitis. If one is to study successfully all that concerns the diagnosis and the treatment, it is important to separate carefully the two states I have just mentioned, chronic and acute appendicitis.

In order to explain things more clearly, it is now classical to establish a comparison between appendicitis and inflammation of the throat; for we know that the initial lesion of the appendix, which has an exceedingly well developed lymphatic tissue, is a chronic folliculitis, comparable to that of enlarged tonsils and adenoid growths; we know also that acute inflammation of the appendix varies from a simple hæmorrhagic spot of folliculitis to the gravest gangrenous lymphangitis, states which may be compared with more or less violent attacks of acute tonsillitis or adenoiditis. And, going farther in the comparison, I am one of those who assert that if, in cases of this kind, the appropriate treatment is surgical, in acute attacks it is prudent, although for other reasons, to let the inflammatory mischief subside before using the knife.

My first care will be now to explain by what symptoms chronic appendicitis reveals itself to us, and what are its diagnostic signs.

It not infrequently happens that during post-mortem examinations, or during various laparotomies, we find evident lesions of the appendix on patients who had never been suspected of appendicitis. But perhaps these cases, whose local symptomatology is really *nil*, would not be so wanting in symptoms as they appear to be if we attributed their dyspeptic attacks to their true cause—that is to say, to chronic appendicitis.

In its common form appendix dyspepsia is marked firstly by gastro-intestinal atony, the abdomen being frequently flatulent, by difficulty in digestion, by eructations and often by obstinate constipation, interrupted sometimes by diarrhœal discharges, which may be caused by a particular kind of food, especially by butcher's meat, fats, and indigestible articles of diet. I have known some patients of mine who could not bear even milk and eggs, and whom I was obliged to feed with vegetables only.

These patients have, as a rule, a coated tongue, offensive breath, and even a subicteric, greyish complexion, a capricious appetite, and habitual nausea. It is not rare for them to suffer also from gastralgia or stomach-ache, coming on without known cause, and in very variable frequency, intensity, and duration. The patient feels a sudden but keen though not well localised abdominal pain, associated with an unexpected pallor and even a fainting sensation. It is easily understood, then, that in certain serious but exceptional cases the growth of children may be stopped by such digestive disorders, and a real cachexy be established. Certain cases who have attended their doctors during years for an obstinate dyspepsia, even for supposed intestinal and peritoneal tuberculosis, or, in adults, for supposed intestinal or stomachic carcinoma, were really suffering from chronic appendicitis.

However latent the local symptomatology of appendicitis may be, it is a rule that a weight, an indefinite pain, or at least an uneasiness draws attention to the right iliac fossa, and we feel by careful palpation, under a normally depressible abdominal wall, the cæcum a little thickened, and without flexibility, distended with gas and gurgling, and sometimes also a small tumour rolls under the fingers, due either to the appendix itself or to enlarged lymphatic glands, although nothing enables us to determine this point with certainty.

By deep palpation, specially on McBurney's point, a slight pain is awakened, which one is made aware of either by the patient's testimony or by a small defensive contracture of the muscles of the abdominal wall. These local signs suggest to us the ileo-cæcal origin of the abdominal disturbance; they exclude, for instance, the idea of nephritic or hepatic colic, but they may lead to error, for they may be the signs of a commencing tuberculous disease of the cæcum and not of appendicitis. We must bear in mind this rare tuberculous lesion, puzzling as to its diagnosis, because it may commence with an acute attack, and even by an iliac abscess. Once I was thus mistaken, when I thought to operate on a simple appendicitis after subsidence of the acute attack; on a second occasion I saw another surgeon's patient, for whom he had opened an acute abscess which remained fistulous. I must admit that I cannot give precise directions for its diagnosis—at least, in those cases where nothing else of a tuberculous nature exists either in the abdomen or elsewhere, even when cæcal infiltration is advanced.

Until now I have spoken of a symptomatology which is always obscure. There are more marked cases manifested by special and paroxysmal phenomena, amongst which vomiting in a variety of forms seems to hold the first rank.

We may observe, firstly, children who have in the morning only frequent gastric vomiting, rarely bilious, and without any fever. In certain cases the vomiting becomes habitual, and to such an extent as to be a grave hindrance to feeding. I remember some children relatively young—between three and five years of age—who, after an attack whose acuteness and form was very variable, remained unable to bear any other food than vegetable broth.

Sometimes there are very different symptoms, which occasion real difficulties of diagnosis. I remember a girl who suffered from obstinate constipation, headache, and retraction of her abdomen "en bateau," so that one of my colleagues diagnosed tubercular meningitis. Another patient, a boy, on the other hand, was constipated, with pronounced meteorism, and another patient had a very acute intestinal obstruction, with complete stoppage of gas and fæcal vomiting, without fever. However, the last had only a paralytic reflex obstruction, and two hours after the opiates employed provoked the emission of gas and the end of every symptom. These patients recovered after removal of the appendix.

In the main, these cases constitute a series of examples where the symptoms of dyspepsia with gastro-intestinal flatulence are exaggerated, either continually or by sudden attacks. The study of the temperature and the pulse prove that infectious phenomena are nearly *nil*. In fact, when we operate we find sometimes some old peritoneal adhesions; most often we find none, and the appearances are those of simple chronic appendicitis. One can only see on the mucous membrane the hæmorrhagic staining of one or two points of folliculitis.

In another series the infectious element becomes more important, and a very frequent clinical form is the one where, although the initial dyspepsia may be very light, or even absent, there suddenly breaks out every sign of an "embarras gastrique febrile"; sometimes after too plentiful a meal, sometimes without any appreciable cause, the child has indigestion, with vomiting, and very quickly the fever runs up to 39° or 40° C. The tongue becomes coated, the breath has a sour smell. At the same time there is abdominal pain, which is localised either around the navel or in the right iliac fossa, and in palpating this latter region you find some pain at McBurney's point, a slight muscular resistance, but no trace of phlegmonous infiltration.

These last objective signs allow us to establish a precise diagnosis if one is called at the very moment of this slight attack, and thus we have learnt the great clinical value of "embarras gastriques" irregularly repeated in the previous history of patients suspected or duly convicted of chronic appendicitis. Beware of those frequent indigestions, which mothers treat with a dose of medicine, when with the sickness are associated sudden fever, and colic principally on the right side.

These attacks, even when not attended to, do not last more than one or two days. But in some cases they grow worse and more frequent, so as to present the classical appearances of the periodical acetonemic vomitings of childhood. I have pointed out facts of that sort and spoken of children attended during several years for such a disease by eminent doctors, and cured only by the removal of their chronically inflamed appendices, and I said that for a patient of that kind it was very important to pay serious attention to the spontaneous pain, or to the one excited by pressure over the right iliac fossa. I said also that for those patients it was necessary to search for the physical signs of chronic appendicitis, such as I have just described them. We shall acquire, in thus doing, the elements of a sure diagnosis which is sometimes neglected. But I have not said or thought anything more on this aspect of the case, and I have been very surprised to read in some recent papers on the matter that I always considered periodical acetonemic vomitings as a form of appendicitis.

When these patients are operated upon, it will be found that appendix or periappendix inflammations are, in the average, more intense than in those of the first series, which have only dyspepsia with apyrexia or little pyrexia. And here, with the ordinary folliculitis associated with an old sclerosis, we find sometimes omental adhesions; but one may say that, with some exceptions, these changes are not worthy of immediate surgical interference, however alarming may be the symptoms. The impression produced in these cases, however paradoxical the assertion may sound, is that of an acute attack associated with chronic pathological changes, and certainly in these cases post-mortem examination will never cause the surgeon to repent of not having operated rapidly enough to anticipate the infection. Besides, if we examine thoroughly these patients' pulse, temperature, functional indications of general or peritoneal infection; if we ascertain, at the same time, the depressibility of the ileo-cæcal region under palpation, we do not find in these signs cause for an immediate operation.

However, I shall not assert that the surgeon is never to interfere in these cases. Rather, on the other hand, I think that it is to the surgeon only that these patients should go, and it is convenient to here state the opinion I shall soon fight for in another sense—"that there is no medical treatment of appendicitis." I admit that patients who suffer from chronic appendicitis and enjoy uninterrupted rest and a most exclusively vegetable diet may, if not cured, have no acute crisis, nothing save some insignificant uneasiness.

But if that rest can be obtained for grown-up patients, exception being made for the working class, it is impossible to obtain it for children, especially in the case of boys, in whom severe exercise, fatigue, a visit to the "tuck-shop"—and often some unknown cause—may readily incite grave attacks by which life is suddenly threatened from peritoneal or even general infection. And then is to be considered the grave question: Must the surgeon operate during the acute crisis?

In order to take, I don't say an absolute and final, but a reasonable step, we must first consider the dangers we have to deal with.

When an acute attack occurs in an appendix, chronically irritated or not beforehand, the initial lesion seems to be the same as I have described previously—that is to say, a more or less intense, more or less limited, folliculitis. But from it may result a real lymphangitis, grave and diffuse, simple or gangrenous, perforating or not. Then intervene in the highest degree the phenomena which had remained at the second stage in the preceding forms—general infection, peritonitis.

General infection seems to be conveyed especially through the veins. The origin of some cases of pyæmia with predominant hepatic abscesses is without doubt a pylephlebitis starting from the vermiform appendix. In other cases there is a true septicæmia without any suppuration of the liver, which, however, undergoes an extraordinarily rapid fatty degeneration; and it must be noticed that, if the appendix is generally found gangrenous in these conditions, that alteration is not indispensable. Lesions are found which are sometimes so insignificant that after operation we have some reason to think that in reality we are dealing with a septicæmia originally intestinal, where the appendix is certainly infected to a certain degree, but is involved only for a part, and even then for a very small part.

These forms, which are not very well known to us yet, are anatomically and clinically very different from those in which complications special to appendicitis depend upon the particular anatomical connections between the appendix and peritoneum. These two principal forms are to be considered according as the peritoneal inflammation is or is not circumscribed by adhesions, for the diffuse peritonitis, more or less septic, and the encysted peritonitis, ending by a localised abscess, are evidently of very different prognosis.

The gravity of these general or local infecting conditions is extremely variable. They stand between the slight attack with very sudden, and transient, high fever, and those in which death occurs in two or three days. And it is easy to understand that these catastrophes haunt our memory; the more because, we must avow it, the beginning of the illness does not always allow us to diagnose with precision what will be its ulterior evolution.

Hence arose the tempting, because simple opinion, that the surgeon must always operate in acute appendicitis as soon as he is called to give his advice. Twelve years ago it was the opinion of almost all surgeons. I don't deny to have shared it, and I am convinced that in acting as we did at the beginning of our practice, we saved numberless lives. It was at a time when our surgical activity was checked by numerous physicians, staunch defenders of medical therapeutics. When we analysed the results, the average of various operators' statistics, we had a mortality of about 25 per cent., and in spite of some medical statistics published at the time, I am persuaded that our success was infinitely superior to that of our opponents. As they have become less and less numerous, it is useless to refute them now.

However, their assertions were partly true sometimes. Very often, after refusing our counsel for immediate operation, they obtained the resolution of what was formerly called perityphlitis and which is now named appendicitis; and, on our part, we became aware that, for patients who had their attack calmed by medical treatment, the operation made in the interval of attacks, "à froid," as we say in France, was very much better. It allows us to remove with ease the appendix, which is not always possible amongst the adhesions of an encysted peritoneal abscess. It almost always enables us to obtain primary union after careful suture, and so we avoid the hernia which is so frequent after the incision of an abscess. And it not only saves the patients from these two causes of a second operation, more or less delayed, but further reduces the mortality to 10 per cent. instead of 25 per cent. in the hands of the same surgeons. We set aside, of course, the operation for chronic appendicitis in which the mortality is almost *nil.* I am speaking now specially of the acute cases that are brought to the hospital, including those we see for the first time when they are already suffering from diffuse peritonitis and almost hopeless. I insist on that point, because there is an important difference between private and hospital practice. In the acute attacks, with localised suppuration, we are liable to inoculate with the pus the adjacent peritoneum not protected by adhesions. Then follows a fatal peritonitis, and now we know that a too early operation was the cause of this diffusion.

So a general understanding was arrived at, or nearly so, between the fellows of our Parisian Surgical Society, and the debate which took place in 1903 proved that almost all our colleagues, renouncing the rule they had adopted in 1899, had given up the practice of always operating immediately. They think now, and it has been my opinion since 1896, that the rule must be: operate immediately if you are called within the first twenty-four hours following the outburst of the acute attack; but when you are called afterwards, you must act according to the indications given by each individual case, and then with the idea of calming as much as possible the attack before operating.

It is certain that the early operations, in the first twenty-four hours, before perforation or gangrene, before adhesions and suppuration, can be performed in anatomical conditions almost identical with those of the operation "à froid," and nearly all the objections previously mentioned are then of no account. But, in order to act in this way, we must possess an absolutely certain diagnosis; and in children this is difficult to obtain, in association with certain clinical features understood by all children's doctors and surgeons, and this is the reason why they came to a general agreement about that subject.

The causes of error exist both for peritoneal and septic forms. Suppose a child presents the symptoms of an acute peritonitis, it is often more difficult to decide quickly in him than in an adult whether the appendix is attacked. The tubo-ovarian inflammation conduces to error, without doubt, in women, but less than pneumococcic and gonococcic peritonitis in childhood; and we must remark that in a great majority of cases the pneumococcic form attacks girls, and that the gonococcic cases are exclusively confined to them. Diagnosis is generally easy when pain in the right iliac fossa is definite, localised, and acute, spontaneous or excited by pressure, and when in a few hours phlegmonous swelling with muscular defence of the abdominal wall is evident. But we must take care and be cautious when children say their abdominal pain is round the navel, and when the swelling is relatively slow in forming. Besides that, pain limited to pressure with muscular defence may also exist in other forms of peritonitis which I mentioned, for pneumococcic peritonitis is usually more or less encysted around the initial point, and the gonococcic starts from a tube, without, however, any tubo-ovarian tumour which could be found by rectal investigation, such as occurs in women.

In gonococcic peritonitis the appearance soon becomes very grave, with a very small and very weak and frequent pulse, with cyanosis of the face, and those obvious local symptoms which will attract your attention if the girl be suffering from vulvo-vaginitis. But at the commencement, save in the rather uncommon case of a previous pleuropneumonia, the nature of pneumococcic peritonitis escapes us entirely up to the moment when pus is collected in the abdomen. As a rule, immediate diagnosis is impossible. On the other hand, it is certain that the opening of the peritoneum aggravates in a disastrous way the virulence of the gonococcic infection ; it aggravates just as much the pneumococcic one in an early stage, before the formation of pus. The first of these two forms of peritonitis will never become surgical; the second one will, but not in the initial stage. In fact, in both forms one may meet with serious complications if one tries the method of exploring laparotomy. On the other hand, laparotomy does not cause any injury in the beginning of tubercular peritonitis simulating the beginning of appendicitis.

Another difficulty arises in children when symptoms closely resembling those of appendicitis, without iliac swelling, but accompanied with sudden fever and vomiting, are due to intestinal helminthiasis. In some of them the symptoms cease after one or two days by the spontaneous evacuation of the ascarides. As early as 1893 I had one of these cases which was published by my pupil Jacob in his inaugural dissertation. These cases have been used to establish the theory of appendicitis provoked by intestinal worms ; I believe that is a false one, but such facts must put us on our guard against operating when we are not able to make with precision an immediate diagnosis.

Certain septic conditions, without any peritoneal symptomatology, but with pain at McBurney's point, also give rise to mistakes. For instance, I see occasionally children who are supposed to be attacked with appendicitis, and who are suffering in reality from pneumonia with an abdominal stitch. This condition is easily diagnosed if the classic stethoscopical signs exist at the same time. But the perplexity is great in the case of a central pneumonia, where the signs become manifest only after two or three days. These cases are not rare enough in children to give us a right to neglect them. Similar conditions are met with in association with pleurisy.

Lastly one must bear in mind, still more, I believe, in children than in adults, the possibility of typhoid fever beginning suddenly, with vomiting, iliac pains and even enlargement of the cæcum and its lymphatic glands. I observed two cases, particularly obscure, where typhoid fever began in this way, though I knew that they were undoubtedly suffering from a previous chronic appendicitis.

My personal opinion, and that of all my Parisian colleagues, children's physicians, and surgeons, is that these unavoidable difficulties in diagnosis do not permit us to adopt the rule (excellent in cases where the diagnosis is certain) of operating in every case within the first twenty-four hours. Again, we are ordinarily not called so soon; in small and slight attacks, which are by far the more common, mothers are accustomed to treat them as an indigestion, and when the doctor is summoned some time has been already lost, and still more is wasted before the surgeon arrives.

The opinion which begins to prevail is the following: that when, for one of the reasons stated, we have decided to wait, we must contine to do so, whilst studying from the moment we are summoned the clinical indications for an immediate, early, or postponed operation, in each particular case.

I think it useless to insist upon the clinical phenomena which require, in diffuse peritoneal forms, immediate surgical interference. Disassociation between pulse and temperature, green vomiting, anuria, dryness of the tongue, alteration of the facial expression, suppression of gas through the anus, are all signs of diffused peritonitis, or of peritonitis near being diffused. We must exclude, however, patients brought to the hospital who present these symptoms after a wearying journey, and who appear much better after some hours' rest.

In these forms of diffuse peritonitis some surgeons, thinking the condition of the patient hopeless, decline to operate and allow him to die. I believe they are wrong, for now and then some patients judged hopeless have been cured by laparotomy. One might argue, perhaps, that peritonitis was not in such cases truly generalised. But the clinical fact remains that when the symptoms of generalised peritonitis exist surgical cure is possible, though exceptional. We must then operate, without regard to keeping up brilliant statistics.

As to encysted peritoneal forms, they must be operated on immediately only if an abscess has collected; in that case immediate operation is followed by remarkable success, and we must not delay it if after the third or fourth day the fever persists and runs high, and if swelling is discovered in the iliac fossa or in the rectum.

Further, without agreeing with the few who advise waiting for fluctuation, we must understand one another about such cases. When in operating we find pus, we must not conclude that the operation could not have been postponed until the day after, as has been said by many observers. For instance, very often in operating a long time after the calmed attack we discover around an appendix sometimes calculous or even perforated remains of an abscess and caseous matter between the adhesions; surely in these cases there has existed pus which has been absorbed.

That being so, I have not been very surprised that, in spite of the scientific precision of a laboratory's investigation, the study of leucocytosis of the blood has led several observers to contradictory conclusions. We have in this one more indication of value; but we have not in it the positive criterion desired by some surgeons, and we must be satisfied to place its diagnostic indications along with the whole of the physical and functional phenomena of which I am going to speak, in studying the cases in which we attempt to calm the attack. In doubtful cases it is unnecessary to say that we must operate more quickly, but we must inquire carefully how the medical treatment has been applied during the preceding days. In fact, we are sometimes astonished how rapidly this treatment, when well managed, acts on patients aggravated by the use of purgatives and by inappropriate feeding.

I consider it useless to give here particulars of what is meant by "medical treatment" in the acute attack. It may be recapitulated in few words: complete deprivation of food, only a few spoonfuls of pure water, and large bags of ice on the abdomen. In former times I always prescribed opium; now I give it only when specially required, when colic is very painful and not explained by an increase of the iliac swelling. I must draw specially your attention to purgatives being dangerous and remark that patients who drink milk or broth do not keep strict diet.

Patients thus treated must be carefully watched so that they may be operated upon, not only when the slightest appearance of peritonitis is noticed, but also if the fever does not yield quickly and if the local swelling progresses.

From all this it results that we should delay less when there is pelvic localisation of the swelling, a form difficult to watch, and of which the diagnosis is made by paying attention to some special clinical facts, such as vesical pain in passing water, and above all by searching systematically *per rectum* with the finger. We must also remember that we are not to act in the same manner with patients who live in town as with those who live in the country: these latter require to be operated on almost always immediately.

According to all these preceding facts, there is no absolutely established rule. But an accomplished clinical man will but rarely allow himself to be surprised by the development of peritonitis during an attack of appendicitis with local swelling, in which he watches an abscess in formation. Disappointments will be less frequent than from diffuse peritonitis following an operation undertaken too late or from one performed too soon, after the beginning of a peri-appendicular abscess, and before the protecting adhesions have formed between the deeply seated pus and the anterior abdominal wall.

The real difficulties of diagnosis and the painful surprises for surgeons are not due to these encysted peritoneal forms, but to certain gangrenous appendices in which diffuse peritonitis, with green or even black vomiting (without doubt the final phenomena) occur, but in which, until that sudden and final outburst, peritoneal symptoms remain unheeded. The patient is dangerously infected, highly feverish, pale-faced, sometimes looking subicteric, with cyanotic lips, but in the abdomen we do not find clear signs. The initial iliac pain draws attention to the appendix, but I have not found it from the beginning specially intense. In handling the abdomen, we cannot detect any swelling and we cause often little pain ; even the abdominal wall can be sometimes easily depressed on all parts when the appendix is in its normal place, and when it is hidden far away from our fingers, in such a region as the top of the pelvis or in the sacral fossa, the absence of physical signs is what one might expect. During the operation we learn why no swelling was felt, as then we find around the gangrenous appendix very little pus and but very few soft adhesions.

Every one of us has cured some of these last patients by operating upon them immediately after the diagnosis was made. But all of us also have, with some others, overlooked the diagnosis during a certain period. Firstly, because, these cases being of a kind in which positive local signs are missing, we remain hesitating about the appendix origin of the observed septicæmia ; secondly, because, the appendix being incriminated, it is very difficult to establish an early diagnosis between the gangrenous form which requires immediate excision of the appendix, and some other very septic forms in which macroscopic alterations of the appendix are very slight, and where it seems that the shock of laparotomy and the disorders caused by anæsthesia are prejudicial in stopping, even for a short time, the hepatic and renal functions already gravely involved by the sudden septicæmia.

There are cases of grave jaundice with blood-vomitings, in which yellow atrophy of the liver seems to be the result of an appendix folliculitis, at first sight insignificant: the operation, even when made at the beginning, as soon as our attention has been aroused only by a slight subicteric shade, does not improve the progress of the disease. Now, I saw two of these cases—in grown-up patients, I must say—with jaundice, fever, and rigors, that improved little by little, so that some weeks after I removed a sclerosed appendix with traces of old folliculitis, but without any trace of peritonitis around it.

Therefore, especially in these cases, we remain doubting, and sometimes we allow death to ensue while we are waiting. But while in peritonitis diffused under these conditions we may accuse ourselves of having lost the opportune moment, there is nothing to demonstrate that in operating earlier we should have succeeded better. Experimental as well as clinical study teaches us that the most rapid excision of an inoculated point does not stop infection if it is of a certain degree of virulence. The more so if we are aware of the inoculation only by total and general reaction in connection with it, especially when so deeply situated an organ as the appendix is concerned.

Those cases are happily very rare. The common cases are the peritoneal ones. For these, well treated, sometimes, I repeat it, an abscess will cause us to perform an immediate operation; rarely, we shall be surprised by diffuse peritonitis if the patient is carefully watched; generally we shall succeed in calming the attack. It is well, then, to remove the appendix, about a month later if the attack has been slight. When the attack has been grave, we must wait longer, according to the case.

When I began to practise, I thought with many of my colleagues that the operation was not necessary after a first slight attack. Now, on the contrary, we have all agreed about the necessity of operating after such a warning. Relapse is not a necessity, but it is frequent, and we cannot guess how intense might be a second attack.

For this reason we are right in saying that there is no medical treatment of appendicitis. When we do not operate immediately it is but to give time for the acute inflammation to subside; then the operation is less dangerous. I always said so, and I insist upon that, because some authors, whose only excuse might be their perfect ignorance of my writings, have described me as an obstinate abstentionist. You will also kindly allow me, in order to end this debate, to quote the phrase I printed in 1900: "The true question to decide is whether non-operative treatment is capable of calming an attack, when well instituted and in time, and if it is not better to try this treatment as often as possible, and to postpone the operation for a time. To that question I answer in the affirmative."

Before closing this lecture, I must indicate the technique I think the best for the operation. This account will be short because opinions agree, save for some details.

(1) Diffuse peritonitis requires firstly a long incision in the right iliac fossa, through which I think we should always try to remove the appendix. Almost always we must add to that first incision a median one, and often a third one, in the left iliac fossa. The drainage must be very free, with very large rubber tubes going in different directions into the abdomen and the pelvis. Sometimes I use irrigations, with boiled water or physiological salt solution, but I do not use them regularly because I am not at all convinced that they improve the always lamentable prognosis of the disease.

(2) Encysted suppurating peritonitis is to be treated as a single abscess by a large opening. No rule need be given: the incision is to be made where the abscess is situated, on the right or on the left, in front or behind. There is a doubt about pelvic abscesses. Some authors teach that the best plan is to open them by rectal incision. It cannot be denied that these abscesses after abdominal incisions progress often worse than the other ones. On the other hand, they sometimes heal spontaneously by discharging through the rectum, hence came the idea to imitate that natural method of evacuation.

In spite of some successes due to that proceeding, I do not believe it ought to become general. I think it is dangerous for the abscesses situated high up, which we must search for blindly, with special trocars; and as to the abscesses low down, which can be felt by the finger near the anus, they almost always can be drained through the iliac fossa—sometimes through the left one, where the swelling and adhesions may be. It is very rare to find the appendix directed downwards and that an abscess has formed in the pelvis whilst the iliac fossa is entirely free. But when it happens then the rectal incision can be of real help. I remember, for instance, a girl whose iliac fossa I opened. I found the peritoneum here normal, but a hemispherical arch of adhesions transformed the pelvis into a large pus-containing cavity. Judging the escape of pus into the peritoneum to be dangerous, I closed the abdominal wall immediately and opened the abscess by the rectal way. The patient was rapidly cured.

After the spontaneous or surgical opening into the rectum, it is scarcely necessary to say that the appendix is not treated, and frequently it gives no more trouble; not always, however, for twice I have been obliged to remove it afterwards, on account of persistent septicæmia.

The discussion as to what is to be done with the appendix in localised abscesses has to do with abdominal incisions. Some surgeons have advised its removal always at once. They say that it is the only sure way to stop the peritoneal infection around it and, later, to prevent such accidents as fistula, relapsing attacks, etc. But, by breaking the adhesions and by inoculation of the serous cavity, this method may cause some post-operative diffuse peritonitis. In these cases I remove the appendix only when I see it easily under the adhesions, and owing to my experience of these cases having extended, I act in this way more and more often. I tie the appendix and cut it with the "thermocautère": the cæcal wall is much too softened to bear a suture destined to bury the stump.

(3) In the delayed operations, "à froid," as we say,

the *absolute* indication is to remove the appendix, and whatever may have been said, it is always possible, but not always easy. Firstly, the appendix may have in the abdomen various positions scarcely accessible when adhesions exist; secondly, because even when we can easily reach the appendix, some difficulties and dangers may arise from adhesions and from relics of old purulent matter. We must recognise the fact that a positive diagnosis of this state is often impossible. It may happen that before operating we feel in the iliac fossa the cæcum to be scarcely thickened or even painful, yet when operating we find, either behind the ascending colon and near the liver, or in the upper part of the pelvis, an adherent appendix surrounded by caseous matter.

For that reason I gave up the vertical incision known in Paris as "Jalaguier's," by which the appendix is removed through the aponeurotic sheath of the right rectus muscle. This muscular band lies afterwards opposite the sutured aponeurotic incision, and prevents the occurrence of a subsequent hernia. By this method the removal of the appendix is very easy in simple cases; but when we have to seek it deeply, and when the cavity must be drained, we expose ourselves to serious trouble. Therefore, after several attempts, and each time after cases where in operating I found unexpected difficulties, I adopted again the oblique iliac incision, named "Roux's incision." But in this the muscles are divided, and often after very good primary union a hernia is to be feared. Then I adopted finally the proceeding of McBurney, in which, after an oblique incision of the skin, the muscles are not divided, but separated along their fibres. The way to the ileo-cæcal region is very direct; and if we meet with difficulties, with lesions calling for drainage, we can cut the abdominal muscles upwards or downwards, and we have then all the advantages of "Roux's incision."

I have finished now with these questions of technique. I could expound to you a general method, but I am fearful lest I should become wearisome in detailing to you how to bury the stump or to close the abdominal wound. Moreover, I have already kept your attention too long whilst addressing you in an unaccustomed tongue. You will forgive me, I hope, for speaking in English in person owing to my desire to be more in touch with you all instead of entrusting your Secretary with the translation of my lecture and the reading of it. (*Read May the* 18*th*, 1906.)

104. A CASE OF DOUBLE CONGENITAL DISLOCATION OF BOTH PATELLÆ.

By A. P. PARKER.

(Introduced.)

The patient was a male aged 14 years. No history of a similar condition in any other members of the family. Labour normal. Began to walk at two years of age; the knees have seemed to "grow out" during last few years.

This patient was shown to illustrate complete dislocation of both patellæ. The displacement is increased during flexion and not overcome during extension.

Both patellæ are to be felt with their internal borders looking forward and posterior surface inwards, lying on the outer surface of the external condyles of the femur. The internal condyles of the femur are much enlarged, and there is marked genu valgum. Still the patient is able to walk without much difficulty.

The patellæ are smaller than natural, but no radiograph has yet been taken of the case.

(*Exhibited at the Provincial Meeting at Oxford June the* 23*rd*, 1906.)

105. A CASE OF CONGENITAL MALFORMATION OF INTESTINE IN A FULL-TERM FŒTUS.

By A. P. PARKER.

(Introduced.)

In this specimen the condition found was as follows: The stomach is normal in size, but the duodenum is enormously distended. Liver and gall-bladder and bile-ducts normal; at distal end of duodenum, where the duodeno-jejunal flexure usually occurs, there is a complete hiatus of the tube of the gut. The small intestine is to be found commencing blindly, lying in a corkscrew manner with only a small mesentery, attached chiefly over what is the enlarged lower part of the second portion of the duodenum. Between the blind end of the enlarged duodenum and the blind commencement of the jejunum, a small tag of intestine is to be seen, quite shut off from either of these portions of the intestinal tract, and having a mesentery of its own.

There is a well-marked appendix, and the large intestine is patent throughout. What is apparently the transverse colon is found dipping behind the enlarged end of the duodenum at the site of the duodeno-jejunal flexure. The fœtus was sent down to be dissected in the anatomical department, and nothing is known of the case or symptoms. The child was a female.

(*Exhibited at the Provincial Meeting at Oxford June the 23rd,* 1906.)

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106. A CASE OF TUMOUR OF THE PELVIS.

By H. P. SYMONDS.

(Introduced.)

The tumour of the pelvis in this girl, aged 10 years, has been noticed for about twelve months, and has been increasing in size and becoming harder during the last three months.

There is marked dilatation of the veins over the pubes. There is no history of injury. Some pain occurs occasionally on micturition and defæcation.

On examination the tumour is found to occupy the left iliac region as high as the level of the crest and almost to the middle line, and it is firm and hard. There are enlarged glands in Scarpa's triangle.

The tumour is probably an enchondroma.

(*Exhibited at the Provincial Meeting at Oxford June the* 23*rd,* 1906.)

107. CALF RINGWORM.

BY ERNEST MALLAM.

(INTRODUCED.)

O. B—, a boy aged 9 years, contracted ringworm early in May. The intense inflammation in the affected area suggested the probability that the disease was taken from a calf.

A visit to the boy's home—about six miles from Oxford—revealed the fact that one other boy in the school was recovering from kerion, and that diseased calves had been kept in a field adjacent to the playground; whilst there were no ordinary cases of ringworm in the school.

The chief reasons for showing the case, even though cured, were: (1) That it seemed to demonstrate certain features which appear to characterise nearly all cases of calf ringworm when communicated to man, and (2) that one wished to emphasise the prevalence of the disease in the Oxford district.

The exhibitor had had seven patients with this disease under his care within the last two months, and all had presented the same phenomena to a greater or less degree. Two men with the complaint were also shown.

The fungus in some seven cases had been found microscopically, and many had been grown by Miss FitzGerald in beer with agar.

At least two distinct species of the tricophyton had been cultivated—the commoner a white growth. But from one—the most severe case—a black culture was obtained.

It is of interest that in this case the calf itself "died from the disease."

Attempts to grow the ordinary pus cocci had proved negative, though a large diplococcus had been found in some cases.

(*Exhibited at the Provincial Meeting at Oxford June the* 23*rd*, 1906.)

108. A CASE OF LAMELLAR CATARACT.

By P. H. ADAMS.

(Introduced.)

Bertie T—, aged 9 years, the subject of lamellar cataract and ichthyosis. He came up to the hospital because he could not see in the distance, and could not see well for reading. Pupils were equal and active.

Ophthalmoscopic note.—There are large central cataracts in both lenses, which have the situation and size of ordinary lamellar cataracts, but differ in being more "mottled" than most lamellars, and being as dense in the middle as at the periphery. There are no "riders" to be seen.

Teeth are good, and not suggestive of lamellar cataract. Patient is the second of six living children. None of the others have defective sight.

In addition to the above, patient suffers from ichthyosis simplex. This is of interest, as ichthyosis is said to be due to some error of development of the skin. Whether the two diseases may be possibly due to some common cause, or whether their occurring together is a mere coincidence, one cannot say. One would like to know if it has been observed in other children.

(*Exhibited at the Provincial Meeting at Oxford June the 23rd,* 1906.)

109. CULTURES OF TRICHOPHYTON MEGALOSPORON ENDOTHRIX.

By GEORGE PERNET.

Mr. George Pernet demonstrated:

(1) Cultures of *Trichophyton megalosporon endothrix* (on glucose-agar, and potato) from the scalp and finger-nails of a girl, aged 15 years, who had had tinea tonsurans for ten years and tinea unguium for some seven years. Microscopically, Mr. Pernet had found segmented mycelia within and without the stumps from the scalp patches (endo-ectothrix, so called), which could be explained by the long duration, the fungus originally inside hairs having broken through. In the nail scrapings he had found fairly abundant mycelia.

(2) Culture of *Trichophyton megalosporon endothrix* on glucose agar, from a girl whose toe-nails and finger-nails were affected. The disease had been contracted in the far East. Microscopically, Mr. Pernet had found mycelial elements in the scrapings from the finger-nails, and also from the reddened areas over the dorsum of the feet (Tinea tropica). The growth on glucose-agar differed from that obtained in the other case.

(*Exhibited at the Provincial Meeting at Oxford June the 23rd,* 1906.)

110. A VARIETY OF CLINICAL CASES AND PATHOLOGICAL SPECIMENS.

Dr. W. J. Turrell (Oxford) (introduced) exhibited a well-marked case of achondroplasia in a girl.

Dr. E. Mallam (Oxford) (introduced) exhibited a child with enlarged liver and spleen for diagnosis.

Mr. R. H. A. Whitelocke (Oxford) exhibited a girl with double hernia of the ovary whose sister had suffered from the same trouble, and from whom one ovary was removed by operation, and reported at a previous meeting of the Society.*

Professor James Ritchie (Oxford) (introduced) exhibited numerous pathological specimens, viz. cirrhosis of the liver in a child of 10 years; granuloma of the brain with nodules in the lungs; tuberculoma of the brain and tumour of the choroid plexus.

Professor Thompson (Oxford) (introduced) exhibited numerous anatomical specimens: (1) illustrating the development of the thymus at various ages; (2) the sexual differences of the pelvis in the fœtus at birth; (3) the lobulated condition of the kidney at birth; (4) the appearances of the ovary at birth and subsequently.

Mr. Finch (Oxford) (introduced) exhibited a well-marked case of cretinism in a child.

(*Exhibited at the Provincial Meeting at Oxford June the 23rd,* 1906.)

111. NOTES ON A CASE OF ACUTE OSTEOMYELITIS OF THE SPINE IN AN INFANT.

By GEORGE CARPENTER, M.D.

In vol. V of the 'Reports' of our Society will be found a paper on "Acute Osteomyelitis and Periostitis of the

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* *Vide* 'Reports,' vol. ii, pp. 191–195.—[Ed.]

Spine"* by Mr. A. H. Tubby, which was read at the provincial meeting at Sheffield last year, and when dealing with this rare condition he reviewed the literature of the subject. It would appear that the disease has received but little attention in this country, but contributions have been made by Ballance, Lucas, Makins and Abbott, and others. There appear to be several varieties of the affection, varying from periostitis without baring of the bone and attended by perfect recovery to a state which is extremely dangerous to life, where suppuration takes place in the bodies of the vertebræ, the processes are laid bare, and spinal meningitis is apt to occur. The case which I am about to relate is an example of the acute variety, and is of interest owing to the tender age of the child.

Dinah da Costa, aged 1 year and 3 months, was admitted into the North-Eastern Hospital for Children, under my care, on October the 20th, 1904. The mother complained that the child was wasting, and had been very fretful and feverish for the past two weeks. She was a breast-fed baby, and had thrived on the breast until the onset of the present trouble, when she was weaned, but gradually became worse. She had been perfectly well until the present illness. Of nine children, seven were healthy, and one died of "bronchitis." The parents were both healthy. There was no history of tuberculosis in the family.

On admission.—The child looked very ill, she was anæmic and somewhat wasted. The temperature was 101° F., the pulse-rate 140 to the minute, and the respiration-rate 52 to the minute.

Both sides of her chest worked equally well. Cry-resonance and cry-fremitus were diminished over the lower part of the left chest behind. There was some dulness at the extreme left base, and the breath-sounds in this region were very faintly heard. In front and below there was marked tubular breathing. The position and sounds of the heart were natural.

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* 'Reports,' vol. v, pp. 302–313.

There was a general fulness and prominence of the abdomen. In the left loin a large, smooth, rounded, and fluctuating swelling could be made out, moving slightly with respiration and dull on percussion. The mass was very tender, and the child cried out a good deal, making it somewhat difficult to define its exact limits.

The urine was of specific gravity 1010, and contained no abnormal constituents. She passed water naturally.

On October the 21st the temperature was normal, the respiration rate 44 to the minute, and the physical signs were unchanged.

On October the 24th the temperature began to rise again—100·6° F. Chloroform was administered and a thorough abdominal examination made. Nothing abnormal could be felt per rectum, and no definite mass could be made out by abdominal palpation. There was merely a sense of resistance and a feeling of something abnormal in the left lumbar region. The temperature rose to 102° F. a few hours after the examination. The respiration became more rapid and the child cyanosed. She died on October the 26th.

Post-mortem examination.—The left pleura contained about two pints of purulent fluid. The right pleura was thickened. The left lung was collapsed, and its base was adherent to the diaphragm. The *heart* and *pericardium* were normal. In front and to the left of the spinal column, near the first, second, and third lumbar vertebræ, there was a quantity of pus which had separated the anterior common ligament from the vertebræ and occupied a cavity bounded in front by the left kidney, the posterior surface of which was excavated. This cavity communicated through a rounded aperture in the diaphragm with the left pleural cavity. The intervertebral disc between the first and second lumbar vertebræ had been completely destroyed, and the adjacent portions of the anterior surfaces of these vertebræ were eroded. Microscopical examination of the pus showed streptococci and diplococci. No tubercle bacilli could be found. The salient points of this case are :

(1) The extreme rarity of such cases.

(2) The variation in the physical signs: (*a*) Signs simulating psoas abscess, together with signs of a small collection of fluid in the left pleura; (*b*) aggravation of the general symptoms, increase of thoracic signs, and lessening of the physical signs in the abdomen.

(3) The communication of the two abscess cavities.

(4) The nature of the micro-organisms found in the pus.

Discussion.

Mr. A. H. Tubby said Dr. Carpenter had referred to the contribution to the Society's 'Reports' which he (Mr. Tubby) made last year. He regarded the present case as a very instructive and interesting one. All who heard the description given must have been struck by the extreme difficulty in diagnosis, and by the reflection that that particular form of spinal osteomyelitis is most often diagnosed, not in the ward, but in the post-mortem room. There were varying degrees of spinal osteomyelitis, but he would speak more particularly of the severe form. That might be divided, briefly, into two kinds: the neural arch form, and the vertebral body form. The first kind was distinct from the second in one or two important particulars. It was more often accompanied by nervous symptoms, while in Dr. Carpenter's case, when the centrum was affected, the nerve symptoms were absent. Secondly, it was comparatively easy of diagnosis, because the neural arches being near the surface they gave rise to a good deal of œdema, and that œdema is out of all proportion to the amount of pus present. Dr. Carpenter's case invited one or two comments, and a question. What was the cause of the osteomyelitis? In almost all the recorded cases it might be referred to some preceding septic condition, associated either with the mouth or with a suppuration elsewhere. Many of the cases which were recorded were associated with gangrenous or other severe forms of stomatitis, an aspect of affairs which he commended to dental surgeons. With regard to cases of osteomyelitis of the bodies of the vertebræ, the main thing which struck one in the literature of the subject was that it is commonly mistaken for an abdominal condition, and that cavity had been opened in the belief that the case was one of acute intestinal trouble, perhaps with pus in the abdomen. He would have liked to have heard more about the nature of the diplococcus which was present, and it would have been interesting to hear of some culture experiments to elucidate the point.

Mr. R. Clement Lucas said he noticed that Dr. Carpenter's report mentioned that the abscess pressed on the kidney, and that led him to ask whether the author noticed at any time any change in the urine, such as the presence of blood in it. Many years ago he was asked to see a case on account of the child having blood in the urine. A swelling was found on the right side, with evidence of disease of the spine, and he forthwith opened a deep-seated abscess in the lumbar region. As a result, the blood which had been in the urine for some time disappeared. But three months later he had a letter from the doctor intimating that again the urine was tinged with blood, and asking what he suggested. He advised the doctor to examine the other side of the spine for an abscess. One was found, it was opened, and again the blood disappeared from the urine. The symptom was clearly due to the pressure of pus on the renal vein, causing a congested condition of the kidney.

Dr. George Carpenter, in reply, said he was not in a position to say what was the cause of the osteomyelitis. The child's mouth was quite healthy. No catheter specimen of the urine of the baby was obtained, but, judging from the appearance of the napkin, the water was normal. One point he thought would have been very freely criticised, and that was the fact that he had allowed two pints of pus to remain in the pleura without diagnosing the condition. When the child was first examined the physical signs of the chest were very slight, but the patient was sitting up. He subsequently examined the infant under anæsthesia and lying down, and during the examination the tumour apparently disappeared. At the post-mortem he was afraid that it would be found that he had ruptured the abscess into the pleural cavity. But he did not now think so. He thought when the child first came under his notice there was a free communciation, and that during manipulation he had extruded pus from the abdominal cavity into the pleura. A free opening was found at the post-mortem examination, and the passage was large and rounded, and evidently of no recent date.

112. A CASE OF ACUTE ATROTHY OF THE LIVER.

By BERTRAM M. H. ROGERS, M.D.

Cases of this disease are, I believe, very rare in children. We have never had one before at the Bristol Children's Hospital, and I believe none have been reported at the meetings of this Society. Indeed, cases of the disease at all are rare, though curiously when I read some notes before our local Society on this case, another in an adult was read.

The patient, a boy aged 4 years, was a native of Bristol, and one of five children, of which two died in infancy from marasmus. A brother is said to have been an out-patient for splenic anæmia. His only previous illness was measles, which he had in December, 1904. Three months after this he was an out-patient for cough, weakness, and "a yellow tinge on the skin," but the notes say he was quite well till a week ago—that is before June, 1905. He was then observed to be distinctly yellow, and was very sick. He complained of abdominal pain. On June the 1st he became delirious, and in lucid intervals complained that he had headache. The parents said he seemed feverish, but from the account given to the registrar they do not appear to have called in a doctor. There was some irritability of the skin, no rash, sore throat, or coryza. No history could be got of unwholesome food or of the boy having eaten anything. He was admitted on June the 2nd, and when I saw him the following day he was in a state of noisy delirium. This became less marked as the day passed, as he became rapidly more and more collapsed. He was intensely jaundiced with all the usual signs—dry irritable skin, dark urine. Examination of the chest showed nothing; the pulse was 100, irregular and feeble, and the liver could be felt one finger's breadth below the ribs.

The boy died on June the 4th at 1 a.m., with a temperature of 108° F.; a small quantity of urine was obtained, but, unfortunately, not tested for leucin and tyrosin. At the post mortem the heart and lungs were found normal; the liver was about the usual size found in children of this age, weighing 17½ ounces; the capsule was wrinkled and the texture soft, the differentiation of the lobes being indistinct. The substance was of a dull red colour; there were no hæmorrhages. The gall bladder contained bile, and there was no obstruction to the flow of bile. The spleen was large and soft, weighing 4½ ounces. The thymus was enlarged and adherent to the apex of the right lung and pericardium. In a cavity within it was a small quantity of purulent *débris,* the remains of an old abscess.

I do not intend to comment on this case. I am aware it is not quite what we find and read of as happening in adults, but my belief is that had the boy's life been prolonged the liver would have shrunk. Microscopically the liver looked as if all the cells had fallen out in cutting the section, a mere reticulum of fine fibrillar network remaining. As to the cause, I believe we are quite in the dark what causes this rapidly fatal jaundice. I suppose it is a toxine found somewhere, but as yet the *fons et origo mali* has not been discovered.

(*Read at the Provincial Meeting at Oxford June the* 23*rd,* 1906.)

Discussion.

Dr. Leonard Guthrie said that what struck him was the resemblance between the present case and those which had been noted as occurring after chloroform, and, to a less extent, after ether inhalation. He asked whether any smell of acetone was noticed in the breath? Also, was the liver prepared in alcohol? Because alcohol would dissolve any fat in it. He did not think more could be said than that it was probably a case of acute atrophy of very unusual occurrence. He believed Dr. Porter Parkinson recorded a somewhat similar case before the Society a short time ago. It was probably due to some toxic infection, the nature of which was not yet understood.

Mr. George Pernet asked whether there was any evidence of syphilis in Dr. Rogers's case, either congenital or acquired?

Dr. George Carpenter said that in the discussion on "Sudden and Unexpected Death in Children" the previous year he had published under the heading of "Undetermined Causes" a case somewhat similar to that narrated by Dr. Rogers in a girl aged 23 months which he thought was toxæmic. The child, who had been slightly jaundiced for fourteen days, out of sorts, feverish, and constipated, was found to be of a bright yellow colour. Her liver was large and reached nearly to the umbilicus. She then vomited bloody fluid several times, became convulsed, the pulse was imperceptible, and she died the following day with a temperature of 101·8° F. Post-mortem the heart was fatty; the liver was large, coloured partly yellow and partly purple, and microscopically was extremely fatty. The hepatic ducts were pervious and empty. The pancreas was normal. The convoluted tubules of the kidneys were filled with oil. The urine was not albuminous, but deposited rounded granules staining a bright yellow, and leucin and tyrosin were not present. Peyer's patches were much altered, swollen, and of a dark purple colour, those high up on the bowel being precisely in the same condition as those near the lower end of the ileum. The large intestine was normal.*

Dr. BERTRAM ROGERS (Bristol), in reply, said there was no odour of acetone in the breath, and the specimen was not prepared in alcohol, but in formalin. There was no evidence of syphilis; the child did not appear at all like a subject of syphilis. As far as he knew, the mother had had no miscarriages.

113. THE OCCURRENCE OF A CONGENITAL HEART MURMUR IN AN INFANT, AGED FIVE MONTHS, DURING AN ATTACK OF WHOOPING COUGH.

By E. C. WILLIAMS, M.B.

THE whole interest of this case depends on the correctness of the previous history. I saw a child, aged 5 months, who had been ill for at least a week with whooping cough. There was a murmur, continuous through the whole cardiac cycle, heard loudest over the pulmonary area, but also heard over the whole chest, back and front. The heart did not appear to be enlarged, nor was there any cyanosis. The child has now recovered from whooping cough, but the murmur persists unchanged. The child was examined a month before the whooping cough by a medical man, who then detected nothing abnormal with the cardiac sounds. It would appear in this case that, owing to the rise of blood-pressure during the paroxysms, either the foramen ovale or the ductus arteriosus was rendered patent. Parrot noted in 62 cases under two years of age the foramen ovale was only closed completely in 4 cases, and in 187 cases between one month and three years the ductus arteriosus was partially closed in 18 cases.

(*Read at the Provincial Meeting at Oxford June the 23rd,* 1906.)

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* 'Reports of the Society for the Study of Disease in Children,' vol. v, pp. 133, 134.

Discussion.

Dr. Bertram Rogers (Bristol) said Dr. Williams gave him an opportunity of seeing the case with him a few weeks ago, and he could bear out every word which that gentleman had said about the case, although he did not see the case when it had whooping cough. It was the father, a medical man in Bristol, who found the heart (?) normal. There was a loud blowing murmur to be heard all over the heart area. The attack of whooping cough was moderately severe, and there could be no doubt the heart had been strained.

Dr. Edmund Cautley thought the case an extremely interesting one. He would hesitate to accept the statement as to the absence of a murmur beforehand—a father's diagnosis was not always reliable. Next, he did not quite gather whether Dr. Williams was convinced that the murmur was entirely due to a patent foramen ovale. No doubt a cough like whooping cough might make a partially closed foramen ovale patent, and give rise to a murmur; but the description of the murmur seemed to be more like that due to a patent intra-ventricular septum. He doubted whether a patent foramen ovale would produce so loud and extensive a murmur. If the murmur were due simply to a patent foramen ovale, produced by increased tension as the result of cough, one would expect that the murmur would gradually disappear, but there seemed to be no sign of it disappearing. He would like to know what the examination of the heart yielded in the shape of evidence of hypertrophy or dilatation on the right side. An examination by the screen might enable one to ascertain whether there was hypertrophy on the right side of the heart, and that would help the diagnosis. He inclined to the view that previous to the whooping cough there was congenital heart disease, which had become accentuated by the cough. Another possibility was that the cough was not purely whooping cough, but was complicated by bronchitis and broncho-pneumonia, and that there had been some endocarditis set up by microbial infection.

Mr. Howell Evans said he was about to refer to the pars membranacea septi, to which Dr. Cautley had referred. With regard to the clinical signs evidenced by various congenital cardiac lesions, he would not dare to bring forward anything in support of his theory in the presence of so many illustrious physicians, but it had staggered him, as an anatomist, to see throughout medical literature the very frequent reference to patent foramen ovale whenever there was any difficulty with reference to a cardiac sound. The pars membranacea septi—a more delicate structure, and one more liable to be snapped from any excessive force on the right side, especially if the individual had a very strong moderator band—was a point which had been very much neglected. He was therefore pleased to hear it mentioned for the first time by a physician. Moreover he would add that the absence of cyanosis favoured the site of the lesions as ventricular rather than auricular.

Dr. G. A. Sutherland asked whether Mr. Howell Evans had known a case in which the septum had gone "snap," and whether he had been able to prove it post-mortem.

Mr. Howell Evans, in reply to Dr. Sutherland's question, reminded the meeting that he said he hesitated to speak of clinical experience, but anatomically the pars membranacea septi was most likely to go, because the relative muscular development of the short ventricular portion of the heart, in comparison with the elastic distensile portion of the auricle, was small. It was impossible to conceive of the reopening of the ductus arteriosus at so late a date, lastly the absence of a sudden or peristent cyanosis contraindicated all evidence in favour of a foramen ovale having been reopened.

Dr. Leonard Guthrie said he saw no difficulty in accepting the theory that there was probably a patent foramen ovale, or at least a valvular form of that; and he thought it possible the murmur might not have existed until, under some strain, the valve became more patent. It was extremely common to find a valvular foramen ovale when there had been no sign of heart disease before.

Dr. George Carpenter said it was a remarkable fact that the physicians attending the meetings of the Society never agreed on the subject of the bruits that were to be heard in the hearts of patients suffering from heart disease or even in their interpretation when heard, and therefore, following precedent, he was about to express a totally dissimilar opinion to those already given. He gathered from Dr. Williams' description that the murmur was continuous, and if so, he did not see how a patent septum ventriculorum or a patent foramen ovale could produce such a murmur, and a continuous murmur in association with these conditions was outside his experience. He thought the murmur described was due to a patent ductus arteriosus, and further, he agreed with the suggestion that the gentleman who made the first examination might not have detected the bruit, as it was easy to overlook such in a fretful infant.

Dr. C. W. Chapman asked whether Dr. Williams would report further if anything happened to the child.

Dr. E. C. Williams (Bristol), in reply, said he never saw the child before it had whooping cough, and he was asked to see it because of the murmur, which alarmed the father. If the murmur had been present before, even if it had been only one fourth as loud as at present, it could scarcely have failed to attract attention. It could be heard all over the chest. The child did have some broncho-pneumonia, but although he repeatedly examined the heart he could find no increase in the area of dulness. Dr. Rogers had done the same thing with a like result.

114. SOME OBSERVATIONS ON ENLARGED VEINS IN CHILDREN.

By A. G. GIBSON, M.B.

(Introduced.)

A large proportion of children brought up to the out-patient department of any hospital suffer from loss of appetite, wasting, and general debility. Physical signs are, as a rule, few. In many of them, however, veins in various situations are more easily seen than in normal children. The commonest situation is on the front of the chest, but they are found frequently on the back between the shoulder blades, under the chin, and on the temples. The veins are exceedingly small and are flush with the surface of the skin. These venous ramifications are not seen in all thin children, as, for instance, in the wasting that results from a cerebral tumour; hence it is probable that these veins are dilated. That in some there is venous obstruction is shown by the presence of dilated external jugular veins on one or both sides, even in the upright position. On deep inspiration they do not collapse as do jugular veins in other subjects—*e.g.* a patient breathing deeply under an anæsthetic.

Accurate records have been kept of fourteen such cases showing as the cardinal sign dilated jugulars on one or both sides, which do not collapse on inspiration. Of these seven were male and seven female; the age varied from four to ten years; a history of tuberculosis in the family was obtained in eight cases; the symptoms were either lassitude or wasting, or referred to the digestive or respiratory systems, such as frequent attacks of vomiting and diarrhœa and attacks of bronchitis or some form of chest trouble. The left jugular vein was enlarged in fourteen, the right in twelve; veins on the chest were visible in thirteen cases, on the back in two, under the chin in two, on the temples in seven. Downy hair on the back was found in six cases; the retraction murmur of Eustace Smith in ten. Small glands in the neck or elsewhere were present in nine cases. Tuberculous peritonitis was present in two cases, while in a third the signs suggested a similar condition.

Only in one case has it been possible to observe the anatomical condition under which the dilatation of the veins is produced. This was in a case of tuberculous peritonitis in which a dilated left jugular vein had been noticed for some time previous to death. A dissection of the anterior mediastinum shows a ring of small fleshy glands surrounding the left innominate vein. Sections made from one of the glands show a few giant-cell systems and tubercle bacilli. The posterior mediastinum in this case shows several hard, caseating glands with large numbers of tubercle bacilli.

The cases correspond in character to those described by Eustace Smith as being due to tuberculosis of the mediastinal lymph glands, and without implying that dilated jugular veins which do not collapse on inspiration are pathognomonic of such a condition, it is suggested that in the presence of a tuberculous history, symptoms of debility, small glands in the neck, veins on the chest, back, neck, or temples, a retraction murmur, and in the absence of signs pointing to syphilis, lymph adenoma or lymphosarcoma, the sign may be of value in the diagnosis of early tuberculosis.

(*Read at the Provincial Meeting at Oxford June the 23rd,* 1906.)

Discussion.

Mr. Clement Lucas said that as a result of these observations one might regard enlarged veins in the neck as generally indicative of tubercular glands; that seemed to be the result of Mr. Gibson's researches. But there were other local causes which he (Mr. Lucas) had seen cause enlarged veins in the neck, one of which was a supernumerary rib.

Dr. G. A. Sutherland said the paper was a very interesting one. Mr. Gibson had been working at the subject, and was able to give direct evidence of a condition of affairs which most had assumed to be present, but had not had an opportunity of determining. Mr. Gibson had directed his attention specially to the anterior part of the neck, and he would like to know whether he had made any observations on the posterior part of the neck. That was a region which he (Dr. Sutherland) had been watching for some time with interest, because one frequently found opposite the seventh cervical spine a considerable dilatation, not so much of the veins as of the venules there. The question which had been in his mind was whether that lesion was due to the presence of glands in the neighbourhood. It was often present in connection with adenoid trouble. Apart from that explanation, which he had not been able to work out thoroughly, he did not know of any reason why that special spot at the back of the neck should be the site of a very marked dilatation of veins or venules.

Dr. Edmund Cautley congratulated Mr. Gibson on his very able and accurate paper, and on bringing forward definite proof of what was supposed to exist before. He called the attention of the meeting to the fact that some years ago a condition was described by a French observer as "venous dystrophy." This was a condition in which the veins of the scalp were very prominent in young children. It seemed very probable from the present observations that this venous dystrophy was due to pressure by enlarged glands, although the French observer ascribed it to congenital syphilis. Dr. A. Morison showed a case at the Harveian Society some time ago, and in that there was no evidence of congenital syphilis. One might conclude from the paper that there was no doubt that enlarged glands would cause dilatation of veins, but, unfortunately, one could not conclude, in the absence of dilatation, that there were no enlarged glands.

Mr. A. G. Gibson (Oxford), in reply, said he did not wish to imply that dilated jugulars necessarily meant enlarged glands in the chest; only that in those cases of which he had notes and had an opportunity of examining, he thought, from the history and other signs, that the cause was the slight enlargement of tuberculous glands. He had not seen a case in which a supernumerary rib had caused dilatation of veins, but that was one of many causes which might produce it. With regard to the posterior part of the neck, he thought the venules which he had observed on the back and on the neck were exactly the same as described. It was not a dilatation of large veins, but a dilatation or greater prominence of the venules. But he had not directed his attention to that part until he had observed several cases, so his notes were somewhat incomplete. In his later cases there were many having dilated venules on the back at the same time as the dilated jugulars, which did not collapse on inspiration. He thanked Dr. Cautley for mentioning the venous dystrophy of the French writer; he had not yet done more than superficially examine the literature of the subject, but, with Dr. Cautley's help, he hoped to do so more thoroughly.

115. NOTES ON A CASE OF INTUSSUSCEPTION.

By HENRY P. CROLY.

(Introduced.)

James G—, aged 9 months, always strong and healthy, was suddenly taken ill with pain and vomiting and diarrhœa at 12 o'clock on the morning of October the 8th, 1905.

The pain continued on and off during the afternoon, and also diarrhœa; at 5 o'clock blood-stained mucus was passed, which continued till I was sent for at 6 o'clock. The child was sitting in its mother's lap on my arrival, not collapsed, and did not appear to be in pain.

The abdomen was very rigid, and it was impossible to make out any definite sign till a few whiffs of chloroform were given, and then an indefinite lump was felt in region of the ascending colon. Nothing abnormal *per rectum*. The child was sent to the Radcliffe Infirmary without delay with the diagnosis of acute intussusception, and was seen by the house-surgeon and Mr. Whitelocke, who confirmed the diagnosis and operated immediately. An incision $2\frac{1}{2}$ inches long was made, having its centre at the level of the umbilicus on the right of the middle line. On drawing out the intestines the intussusception readily came into view. It was of the ilio-cæcal variety. The appendix was bulbous at its extremity and very congested. It was invaginated into the colon to its bulbous end, which, together with the traction of its mesentery, mechanically prevented any more invagination taking place.

It was readily reduced, and the child got over the operation well, and left the hospital three weeks later. The case is interesting as the operation took place eight hours after the first symptom, and it is very unusual to get them operated on as early as this. It also is of interest as the diagnosis was made partly on a sign which Mr. H. L. Barnard first pointed out a year or so ago at the London Hospital. The sign depends on the fact that after a very short time acute intussusception is an intestinal obstruction, and, therefore, when once the lower bowel has been emptied no more bile can pass down, and then there is no yellow stain on the child's diapers; which differentiates it from acute colitis. This sign, and the slight thickening in the region of the ascending colon was all there was to form an opinion on when it was sent into the hospital.

(*Read at the Provincial Meeting at Oxford June the 23rd,* 1906.)

Discussion.

Mr. R. H. A. Whitelocke (Oxford) thanked Mr. Croly for showing the case, for since it left the hospital he had not had an opportunity of seeing it. He attributed the success of the operation in a child so young to the fact that it was seen and diagnosed at once by Mr. Croly, who had to send it in by train twelve miles. Within eight hours after the illness began the child had been operated on and returned to bed. On first seeing the child it was impossible to feel the lump, and not until he was fully under an anæsthetic could it be properly felt. The operation presented very little difficulty. There was already some free fluid in the abdomen. The invagination was released by squeezing from below upwards. Even after so short a time the appendix, which had become more or less bulbous at its tip, was congested and rigid. The process of invagination of the bowel had been arrested by the curling of the appendix upon itself to traction upon its mesentery. The reduction of the intussusception was easy owing to the early stage at which it was met with, and the operation was quickly performed. He did not remove the appendix, as he wished to shorten the time of the operation as much as possible.

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